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# PROCEEDINGS

OF THE

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# SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

**Ninety-third meeting.**

*Cornell University Medical College, October 16, 1918.*

*President Gies in the chair.*

1 (1376)

## Canned tomatoes as an antiscorbutic.

By ALFRED F. HESS and LESTER J. UNGER.

[*From the Bureau of Laboratories, Department of Health.*]

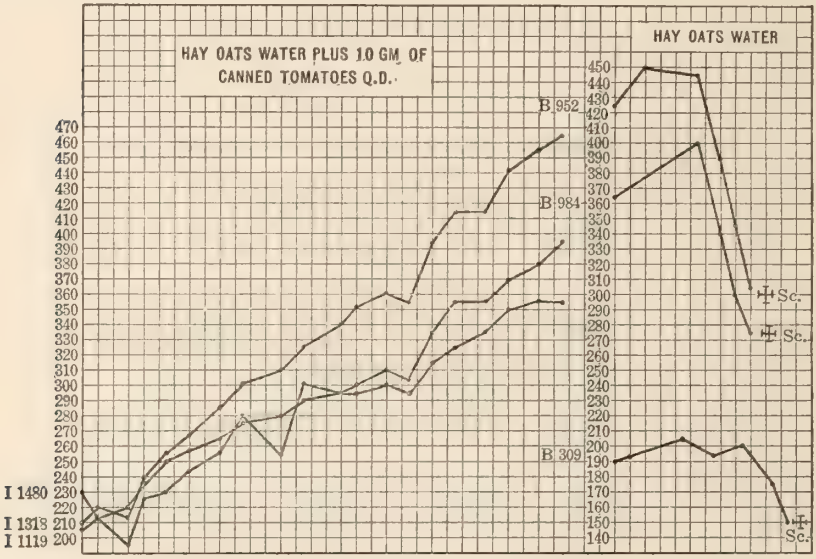
In view of the fact that canned tomatoes are included in our army ration, in which they may be substituted for potatoes to the extent of 20 per cent., it seemed worth while to ascertain whether they possessed antiscorbutic properties. This appeared particularly desirable in view of our previous experiments[1] which had demonstrated that the dehydrated vegetables commonly employed cannot be relied upon for furnishing this important dietary factor. From a theoretical standpoint a study of this question was of further interest, as the tomatoes have been subjected to a temperature above the boiling-point in the course of the canning process.

Series of guinea pigs, five in each group, were put on a diet of hay, oats and water ad libitum and fed various amounts of strained tomatoes which had been canned almost one year previously. It was found that the addition of 5 c.c. of these tomatoes was sufficient to protect the animals from scurvy, and that larger amounts stimulated growth to a remarkable degree.

In view of this favorable experience on animals, for the past three or four months we have fed canned tomatoes to infants who

[1] Hess, Alfred F., and Unger, Lester J., PROC. SOC. EXP. BIOL. AND MED., 1918, XV, p. 141.

were receiving pasteurized milk, substituting it in the dietary for orange juice which has become increasingly expensive. The amount given to babies three months or more of age was 15 c.c.; half this quantity being given daily to younger infants. The




The guinea pigs represented in the graphs on the left received canned tomatoes and thrived. Those on the right were not fed tomatoes and died of scurvy.

tomatoes have been uniformly well tolerated throughout the summer by babies as young as one or two months of age, and we can recommend this foodstuff as an economical and efficient antiscorbutic.

2 (1377)

## Preliminary observations on the value of raw and dried tomatoes as antiscorbutic foods for guinea pigs.

By MAURICE H. GIVENS and HARRY B. MCCLUGAGE.

[*From the Department of Physiology, University of Rochester, Rochester, N. Y.*]

The external signs of the nutritional failure known as experimental scurvy, which can be produced in young guinea pigs in

16–26 days by feeding a mixture consisting of whole soy bean flour, milk, yeast, paper pulp, and inorganic salts,[1] have not been encountered during a period of 75 days in which 10 gm. of *raw* tomatoes were added daily to the diet. If on the appearance of the clinical scorbutic manifestations attributable to the scurvy-producing diet, 10 gm. of raw tomatoes are added as a daily supplement, the symptoms will subside and the animals will be restored to health.

Tomatoes *dried* in a blast of air either for 14–24 hours at 55–60° C. or for 36–44 hours at 35–40° C. retain some of their anti-scorbutic property. This statement is based on the fact that young guinea pigs receiving a daily addition of 1 gm. of either of such dried products have grown and continued in apparently perfect health for a period three times as long as that within which the usual scorbutic symptoms appear.

Further experiments are being conducted upon this subject.

## 3 (1378)

### A method of producing experimental shock.

By **T. S. Githens, I. S. Kleiner, A. L. Meyer and S. J. Meltzer**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

In the numerous investigations on experimental shock there is no uniformity as to the method of inducing it. Some authors simply say "the animal was reduced to shock," without stating by what method it was induced or how long it took to induce it. Others claim that they have produced profound shock by continuous stimulation of sensory nerves, while other writers are quite positive that it is impossible to induce shock by this method. The method *par excellence* of producing shock seems to be the exposure of the abdominal viscera. Here again opinions differ. For instance, Erlanger and his coworkers say that in order to reduce blood pressure to 50 mm. mercury, an exposure and *manipu-*

[1] B. Cohen, Proc. Soc. Exp. Biol. and Med., 1918, XV, 102; M. H. Givens and B. Cohen, ibid., 1918, XV, 126; Cohen, B., and Mendel, L. B., *J. Biol. Chem.*, 1918, XXV, 425; M. H. Givens and B. Cohen, *J. Biol. Chem.*, 1918, XXXVI, 127.

*lation* of the intestines lasting from two to five hours is necessary. In the studies of shock by Githens and Meltzer "the intestines and stomach were dislocated and *frequently handled.*" Wiggers on the other hand states that a more regular and certain circulatory failure is induced *when the intestinal loops are not manipulated.* Many investigators kept the animals under surgical anesthesia throughout the experiment or at least during the greatest part of it.

Generally the fall of blood pressure is the sole criterion of shock. In the studies of Githens and Meltzer the fall of blood pressure and also the disappearance of pain sense were taken as criteria. They studied these phenomena an hour or more after the discontinuation of ether. Of forty-two dogs, in fifteen the blood pressure did not reach a level below 95 mm. within five hours after opening the abdomen, and pain sense returned as soon as the animals came out of ether. In only thirteen dogs the blood pressure reached a level below 70 mm. within two and a half hours and there was no return of pain sense. In these thirteen dogs the original blood pressure was not high. In eight dogs the blood pressure sank to a lower level, but sensation of pain returned when ether was discontinued. In six dogs sensation was lost while blood pressure was still above 95 mm. of mercury.

In experiments undertaken to throw light upon a certain problem in shock, we came across a method which seems to be effective in producing shock in every case and fairly early. The signs of shock were obtained with this method without exception in an unbroken series of experiments on seven dogs and nine cats. The method consists in repeated, strong compression with thumb and finger of the small gut, care being taken to avoid traction upon the mesentery. In all the animals the original blood pressure was fairly high. The strongest effect was a fall of blood pressure from 145 to 45 mm. within one hour and four minutes (cat). In all the animals a considerable fall of blood pressure was obtained within about an hour and fifteen minutes. It seemed that when the compression was produced during deep etherization, the fall began early and was more profound and of longer duration. Whether or not etherization had been deep, loss of pain sense was noted early. In most instances the blood pressure rises moder-

ately again when the animal is left for some time without ether and without squeezing of the gut. In one dog with original blood pressure of 160, the blood pressure which fell to 68 mm. rose after three hours to 120 mm. It is different, however, with the sensory shock. In not a single instance did the pain sense return at any time. This holds good even for cases in which the lid reflex was prompt.

The subject will require a great deal of detailed study. But we thought of putting our experiments on record on account of the value it may have for experimental shock and especially on account of its possible practical importance in human surgery. In abdominal surgery no care is taken to avoid compressing of the gut; on the contrary, it is often employed to achieve a definite end. On the other hand, traction on the mesentery is carefully avoided. In our experience traction on the mesentery rather favors some rise of blood pressure.

4 (1379)

## Metabolism of p-hydroxybenzoic acid and p-hydroxyphenylacetic acid in the monkey.

By CARL P. SHERWIN.

[*From the Chemical Laboratory of Fordham University Medical School.*]

*p*-hydroxy benzoic acid was fed to a monkey in one, two and three gram doses, the urine was collected for 36 hours following each dose, evaporated and extracted. The urine was found in every case to contain only the uncombined acid. This agrees with the findings of other investigators who have fed this acid to several of the lower animals. In each case from 50–60 per cent. of the acid was recovered from the urine.

After feeding *p*-hydroxyphenylacetic acid in one or two gram doses, approximately 60 per cent. of this acid was recovered from the urine of the monkey. Some of this acid existed in the free state, while a portion of it was excreted in the urine in combination with glycocoll as *p*-hydroxyphenaceturic acid. *p*-hydroxy-

phenaceturic acid up to this time was found only on one instance and the amount was insufficient for analysis. The melting point of 154.5–155 is two degrees higher than the melting point cited in the literature. The acid is relatively soluble in alcohol, ethylacetate and warm water but insoluble in ether, benzol and cold water. On boiling with conc. HCl, the acid split up into its two components *p*-hydroxyphenylacetic acid and glycocoll. The analysis of the compound agrees with the theoretical values for carbon, hydrogen and nitrogen.

The process of metabolism in the organisms of the monkey in regard to *p*-hydroxybenzoic acid and *p*-hydroxyphenylacetic acid is comparable to that found in lower animals and unlike that found in man. In man, the *p*-hydroxybenzoic acid is combined with gloycocoll and excreted as *p*-hydroxyhippuric acid, while in the lower animals, the acid after ingestion is excreted uncombined in the urine.

*p*-hydroxyphenylacetic acid on the contrary is found free in the urine of man after ingestion and combined with gylcocoll in the urine of animals.

5 (1380)

## Influence of mere opening the abdomen, of state of shock, and of subsequent section of sciatic nerve upon the blood flow from the femoral vein in cats.

By **T. S. GITHENS, I. S. KLEINER, A. L. MEYER and S. J. MELTZER.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

From a series of experiments performed for the purpose of solving a certain problem in shock, a few definite facts were selected to be put on record. The following results were obtained from experiments on fourteen cats. The rate of the blood flow from the femoral vein shortly before opening the abdomen was taken as the unit, *i. e.*, 100 per cent. The figures indicate the time required for the flow of the same amount of blood, and hence express an inverse ratio to the rate; *e.g.*, 200 per cent. indicates that the rate was half as fast.

*Influence of opening the abdomen.*—The average time of flow from the femoral vein shortly after opening the abdomen in the fourteen cats is 136 per cent., that is, an average decrease of 36 per cent. in the rate. A decrease in rate of flow occurred in every experiment but one. The maximum decrease (in one cat) was 84 per cent. This result means that after the mere exposure of the abdominal viscera (before any change in volume and composition of the blood could have taken place) a slowing of the flow from the extremities takes place. In interpreting this fact the statement of H. Fischer, the first writer on shock from a physiological point of view, is worth considering, namely, that a certain degree of engorgement of the veins of the abdominal viscera can be observed immediately after opening the abdomen. This occurrence may be the cause of the slowing of the flow from the veins of the extremities. As far as we know these facts have not been observed or discussed by any experimental or surgical writers. It may be mentioned that there was a variable but generally a moderate fall of blood pressure immediately after opening the abdomen; but there was no definite proportion between the fall of blood pressure and the decrease in the rate of flow.

*The rate of blood flow in shock.*[1]—When the blood pressure had fallen to about 70 mm. of mercury, the animals were considered in shock; in the majority of cases it fell to 60 mm. or less; the lowest pressure reached was 30 mm. In every cat the blood flow in shock was slower than just after opening the abdomen. On the average it was 301 per cent. of the original. The decrease in the rate of flow seemed in a general way to follow the degree of fall of blood pressure, but was not absolutely proportional.

*Influence of cutting the sciatic nerve upon blood flow in shock.*—After section of the sciatic nerve the rate of blood flow increased markedly in eleven cats, slightly in two and in one there was a slight decrease. The average increase over the flow in shock was about one third, that is, the time of flow decreased from 301 to 196 per cent., but the average rate was still only about half as

[1] In the present series of experiments the blood obtained from the femoral vein was mixed with a small amount of sodium citrate and reinjected through the jugular vein. Hemorrhage therefore was not a factor in the production of shock in these experiments.

fast as the original flow (100 per cent.). In five cats the flow became as fast (or even faster) as immediately after opening the abdomen; and in one of these five the flow was as fast as the original. The blood pressure rose slightly on section of the nerve in eleven cats. The remaining three were those in which there was little or no increase in flow.

The fact that in these experiments the blood flow after section of the sciatic nerve in shock is usually far below normal, and never above normal, certainly does not support the assumption, now current, that vaso-constriction is greater in shock than in normal conditions of the animal.

6 (1381)

## Experimental studies of plant pigments.

By BENJAMIN HARROW and WILLIAM J. GIES.

[*From the Biochemical Laboratory of Columbia University, at the College of Physicians and Surgeons, New York.*]

This communication was confined to a report on studies of (*a*) *flavones*, a group of yellow pigments, characterized by the production, in their solutions, of intense yellowish-brown color on the addition of ammonia, and of (*b*) *anthocyanins*, a group of red, violet, or blue pigments, which, in solution, change to bluish-green on the addition of alkali, and pink on the addition of acid.

These pigments were obtained from tulips: flavone, from "*La Reine*"; anthocyanin, from "*Crimson King.*" Both varieties of flowers were collected at the N. Y. Botanical Garden through the courtesy of Dr. A. B. Stout.

*The chemical relationship of flavones and anthocyanins.*—Wheldale and others believe that flavone is convertible into anthocyanin by *oxidation*. The Armstrongs regard this conversion as due to processes of oxidation *and* reduction. Combes and Willstätter consider that *reduction alone* effects the change. The results of our own experiments accord with the view of the latter investigators. We find that active ("nascent") hydrogen reduces flavone to anthocyanin. The latter can be further reduced

to a leuco-base, which in turn, by exposure to air or, more rapidly, by addition of an oxidizing agent, is reconverted into anthocyanin, thus possibly explaining the facts observed by the Armstrongs:

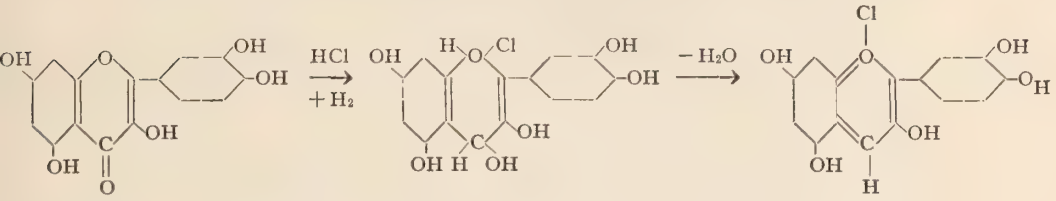
$$\text{Flavone} \xrightarrow{+H} \text{Anthocyanin} \underset{+O}{\overset{+H}{\rightleftarrows}} \text{Leuco-base}$$

We have also succeeded in oxidizing anthocyanin to flavone. This was done most effectively by first isolating anthocyanin in a fairly pure condition, by Willstätter's method. The only oxidizing agent we have been able to use with success thus far is hydrogen peroxide. Oxidase from potato, as well as oxidase from *Crimson King* tulip, could not be used as a substitute for hydrogen peroxide.

Flavone thus obtained from anthocyanin can be reduced back again to anthocyanin:

$$\text{Anthocyanin} \underset{+H}{\overset{+O}{\rightleftarrows}} \text{Flavone}$$

The chemical relationship between flavones and anthocyanins may be expressed thus (Willstätter):




*Anthocyanin as an indicator.*—Anthocyanin (tulip, *Crimson King*), when extracted with absolute alcohol made anhydrous with copper sulfate, yields a *red* extract, whereas if extraction is made with absolute alcohol rendered anhydrous with calcium oxid, a *green* solution is obtained. The red extract can be changed to green by the addition of calcium oxide, or soluble *alkali*, whereas, vice-versa, the green extract can be changed to red by the addition of anhydrous copper sulfate or soluble *acid*.

The extreme delicacy of this reaction naturally suggested the possible use of anthocyanin as an indicator. Anthocyanin, prepared according to Willstätter's method and dissolved in absolute alcohol, was compared with phenolthalein, by the Henderson and Palmer indicator-method for the determination of the concentration of hydrogen ions, with the results tabulated below:

| Tubes | $P_H$ | Phenolthalein | Anthocyanin |
|---|---|---|---|
| 1 | 9.27 | Red + + | Yellowish green + + |
| 2 | 8.70 | Red + | Yellowish green + |
| 3 | 8.00 | Colorless | Colorless |
| 4 | 7.48 | " | Pink + |
| 5 | 7.38 | " | Pink + + |
| 6 | 6.90 | " | Pink + + + |
| 7 | 6.70 | " | Intensity of pink coloration increases in the direction of 11, with increase in acidity |
| 8 | 6.30 | " | |
| 9 | 6.00 | " | |
| 10 | 5.70 | " | |
| 11 | 5.30 | " | |

From these data it is obvious that, in point of delicacy, under the conditions specified, anthrocyanin is, in general, the equal of phenolthalein. Furthermore, anthocyanin is superior to the latter in the fact that a change from alkali to acid is indicated by a sharp change from green to red, and not, as for phenolthalein, from red to no color at all.

The relationship of these observations to those by Gies,[1] on "alkaverdin," will be indicated in a later communication.

7 (1382)

## The probable cause for the failure of some sodium tungstate to give a suitable reagent for the determination of uric acid.

By GRETE EGERER and FRANCES FORD (by invitation).

[*From the Chemical Laboratory, Department of Medicine, Medical School, University of Minnesota, Minneapolis, Minn.*]

In one of his articles, Folin mentions that some preparations of sodium tungstate on the market do not yield a satisfactory

[1] Gies, Chemical studies of the pitcher plant, *Sarracenia purpurea; Journal of the New York Botanical Garden*, 1903, iv, p. 37.

reagent for uric acid, presumably because of impurities such as nitrates and molybdates. The tungstate which we have in our laboratory failed to give the reagent although free from the impurities mentioned above. Further investigations, however, disclosed that our sodium tungstate contained but one third tungstate the rest being sodium carbonate. The reagent was therefore prepared in the following manner:

Dissolve 25 g. of sodium tungstate in 300 c.c. of water, heat to boiling in a large beaker, add 40 c.c. of conc. hydrochloric acid whereupon the tungstic acid is precipitated. Continue to boil for ten minutes. Allow to settle. Filter by suction. Wash precipitate with cold water while still in Buchner funnel, 20 c.c. each time, discontinuing as soon as the precipitate starts to pass through the filter, which usually occurs at the third washing. Transfer to an Erlenmeyer flask without drying the precipitate. Add 15 c.c. of 10 per cent. sodium hydroxide solution for each ten g. of tungstic acid, which will dissolve the tungstic acid on heating.

Continue according to instructions given for the preparation of reagent using but 31.2 g. of tungstic acid in place of 100 g. of sodium tungstate as required in the original description of the reagent.

In our search for the explanation of the failure to obtain the reagent we had to limit ourselves to the use of the two tungstates in stock in our store-room; one of them free from carbonate gave the reagent, the other containing carbonate failed to do so. We are postponing the investigation, to determine if any tungstate free from carbonate will react in the desired manner, until the substance is more accessible. The writers regret that they were also unable to procure tungstic acid itself which they believe could be used in place of the tungstate, by dissolving the acid in a slight excess of sodium hydroxide followed by the procedure given above.

It is necessary to establish the content of actual tungstate only once in order to know how much sodium hydroxide solution has to be added. This is accomplished by transferring the tungstic acid precipitate to a weighed porcelain dish. After drying the contents on a waterbath and subsequent washing with alcohol the dish is weighed again. For preparing the reagent, however, the isolation of the sodium salt is unnecessary.

# SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

**Ninety-fourth meeting.**

*New York Post-Graduate Medical School.*

*President Gies in the chair.*

8 (1383)

## A simple method for making p-arsanilic acid.

By **PHILIP ADOLPH KOBER** and **WALTER S. DAVIS.**

[*From the Division of Laboratories and Research, New York State Department of Health, Albany, N. Y.*]

The preparation of what is now known as primary arsanilic acid, the starting point and the basis of Ehrlich's synthesis of salvarsan, was first mentioned by Béchamp[1] in 1863. In spite of Béchamp's published work, and that of many others, the fact remains that the literature does not contain definite directions for making the primary arsanilic acid. Many laboratories have spent much effort, money and time in trying to evolve the synthesis from the general directions given in the literature, but without much success. The process in its highest form is generally understood to be a trade secret.

Without going into details, it can be shown that the directions either call for too much aniline or too high a temperature, or both. By controlling one or both of these factors one can obtain the primary free from secondary in large yields, practically pure, with ease.

One thousand c.c. of crude arsenic acid (75 per cent.) are heated in an open beaker or vessel, at 120° to 140° C. for 12 to 15 hours by means of an oil or "Crisco" bath. This should concentrate the acid to practically 100 per cent.

[1] *Compt. rend.*, LVI., 1172, 1863.

Fourteen hundred c.c. of dry aniline oil are cooled with an ice mixture to 0° C. or lower, and the cooled arsenic acid is added slowly with vigorous stirring. The mixture soon becomes thick and then granular, after which it is finely ground and thoroughly mixed. This powder has roughly the composition of $(C_6H_5NH_2)_3 - (H_3AsO_4)_2$.

Two hundred grams of this powder are heated in an Erlenmeyer flask, by means of a "Crisco" bath to 160° C., when the powder begins to melt. The substance is stirred continually and when it has all melted, a reflux condenser is then attached; for one and a half hour it is heated from 160°–170°, and then one hour from 180° to 183° C. After allowing the mixture to cool somewhat 225 c.c. of 6N sodium hydroxide and 225 c.c. of water are added, which causes the substance to dissolve and separate out the aniline oil left uncombined.

When the mixture is cool, the aqueous layer is drawn off with the aid of a separatory funnel, and after shaking with 15 to 20 grams of infusorial earth or kaolin, it is filtered with suction.

To the clear filtrate is added 100 c.c. of 6 N hydrochloric acid.

By means of 25 c.c. portions and further additions of 0.5 c.c., 1.00 c.c., 2.00 c.c., etc., of 6 N hydrochloric acid, one finds out if further addition of acid to the whole filtrate will give a larger yield or not. When after waiting a few minutes for the crystallization to take place, and when the best medium has been determined on the small aliquot portion, an equivalent amount of acid is then added to the whole filtrate. Usually the crystals fill the entire solution so that it has the appearance of being solid.

After standing for an hour or longer the precipitate, which is white and crystalline, is filtered with suction. It is then washed by suspending the precipitate in 200 c.c. of water and filtering with suction. When all the wash liquid has drained from the precipitate, it is dried by fanning, or in any other suitable way. The yield is about 30 per cent. of the theory, whereas 10 per cent. seems to be the average heretofore.

The products obtained by the above directions are almost pure enough for further use, but if necessary can be reprecipitated with alkali and acid, or by dissolving it in boiling water and allowing the solution to cool.

The analysis of a crude product once purified by hot water crystallization and dried in a vacuum desiccator gave:

| | As, % | N, % |
|---|---|---|
| Product | 34.20 | 6.02 |
| Product | 34.50 | 6.13 |
| Theory for primary | 34.60 | 6.45 |
| Theory for secondary | 25.65 | 9.65 |

9 (1384)

## Vitamines in green leaves.

By THOMAS B. OSBORNE and LAFAYETTE B. MENDEL.

[*From the Laboratory of the Connecticut Agricultural Experiment Station, and the Sheffield Laboratory of Physiological Chemistry in Yale University, New Haven, Conn.*]

In view of the very scanty data recorded respecting the relative content of vitamine in green foods compared with other plant and animal products which have been studied more in detail, we have fed albino rats on diets containing, as the source of water-soluble vitamine, spinach, cabbage, clover, timothy and alfalfa, dried in their immature state. Far less dried spinach supplies sufficient water-soluble vitamine to promote normal growth than do whole wheat, soy beans, dried egg, meat, milk or potatoes. Spinach leaves are much richer in the fat-soluble vitamine than are most of the products used in our ordinary rations. Thus rats fed for over 160 days, during which they consumed only 25 to 34 grams of spinach, have grown from 60 to 250 grams at a nearly normal rate and have as yet shown no evidence of deficient nutrition. Such quantities are not much larger than have heretofore been considered to be necessary when butter fat supplied the fat-soluble vitamine. A somewhat larger quantity of cabbage than of spinach leaves is needed to promote normal growth. It probably also contains the fat-soluble vitamine. Timothy, clover and alfalfa contain both vitamines, but further experiments are needed to establish the relative amounts of these. Tests of a large variety of tubers, stems, leaves and fruits are now in progress.

If one may draw conclusions from the limited data at present available, it seems that the green vegetables and fodders are richer

in vitamines than most of the staples like meats, potatoes, cereals, fats and sugar products, in the diet of man. Therefore they unquestionably contribute largely to the dietary need of the average person.

10 (1385)

**Preliminary report on the behavior of certain local anesthetics.**

By CARY EGGLESTON and ROBERT A. HATCHER.

[*From the Department of Pharmacology, Cornell University Medical College, New York City, N. Y.*]

The fatal intravenous dose of each of the several substitutes for cocain varies enormously with differences in the rates of injection. Five or more times the fatal dose for sudden injection can be given in a period of one to two hours without causing death. The subcutaneous doses show even wider variations among the different drugs than the intravenous doses.

All of the local anesthetics tested, including cocain, are mutually and quantitatively synergistic. They are all synergistic with epinephrin in its effect upon the blood pressure in a manner analogous to cocain.

The systemic toxic actions of all of the members of the group are very closely alike and all cause death in cats by combined paralysis of the heart and respiratory center.

Three of the members of the group—procain, stovain and apothesine—have been shown to be destroyed rapidly by the liver. All of the others are rapidly destroyed in the animal body, excepting cocain, and it seems probable that this destruction also takes place in the liver.

Artificial respiration alone, or combined with cardiac massage, does not suffice to permit recovery from the sudden intravenous injection of 125 per cent. of the fatal dose of any of the local anesthetics. Artificial respiration and cardiac massage, combined with the intravenous injection of epinephrin, permit recovery in most cases from 125 to 150 per cent. or more of the fatal vein dose of all of the local anesthetics. The previous administration of ouabain permits recovery from 150 per cent. of the fatal dose of

procain when artificial respiration is employed and the similarity in actions of all of the drugs leads one to suppose that it holds true of the other members of the group.

11 (1386)

## A bacteriological report of influenza cases at Presbyterian Hospital.

By MIRIAM OLMSTEAD.

[*From the Presbyterian Hospital, New York City, N. Y.*]

One hundred forty-five cases have been studied in one or more of the following ways: by naso-pharyngeal cultures, blood cultures, sputum for pneumococcus type or lung cultures post-mortem. *B. influenzæ* was found to predominate in the naso-pharyngeal cultures, *Pneumococcus III.* in the blood cultures and *Pneumococcus IV.* in sputum and lung cultures. The percentage of naso-pharyngeal cultures containing *B. influenzæ* was somewhat higher among the uncomplicated cases than among the pneumonia cases. At one period, influenza bacilli were found in 75 per cent. of the naso-pharyngeal cultures. The *Pneumococcus IV.* strains isolated have been found to belong to various groups.

The blood of influenza convalescents has been found to contain agglutinins for strains of influenza bacilli isolated during the recent epidemic. The macroscopic method is used. Incubation at 55° C. gives more satisfactory results than at 37° C.

12 (1387)

## Influence of hydrogen ion concentration upon enzymic activity of three typical amylases.

By H. C. SHERMAN, A. W. THOMAS and M. E. BALDWIN.

[*From the Laboratory of Food Chemistry, Columbia University, New York.*]

Pancreatic and malt amylase and the amylase of *Aspergillus oryzæ* (prepared from taka-diastase) have been selected as representative of the starch-splitting enzymes of the higher animals,

higher plants and fungi respectively. Laboratory methods for the purification of each of these amylases have been described in previous papers. The present experiments were performed with enzyme preparations which had been purified in accordance with these methods. The experiments establish for each of the three amylases the limits of hydrogen ion concentration within which any enzymic activity is shown, and the form of the curve representing the activities at all concentrations of hydrogen ion between these limits. The investigation was carried out with the aid of a grant from the Carnegie Institution of Washington.

13 (1388)

## Studies on the amylolytic activity of human saliva with a new method.

By VICTOR C. MYERS and ANNE G. DELLENBAUGH.

[*From the Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital, New York.*]

Saliva is the one glandular secretion which can be readily obtained in the human subject under relatively constant conditions, and its amylolytic activity has therefore been a time-honored topic of investigation. Nevertheless the methods used for estimating this activity have been rather crude or tedious. A method is described below which is simple, delicate, and, we believe, very accurate. With it a large series of comparable figures may readily be obtained. The method is similar to that which has been employed here in estimating the diastatic activity of the blood.[1]

The technique is as follows: A specimen of mixed saliva, obtained by the stimulation of paraffin chewing, is filtered and a small portion accurately diluted 1 to 100 with distilled water, and also another portion with 0.3 per cent. sodium chloride as an activating solution. After thorough mixing 1 c.c. of the diluted saliva is pipetted into a test-tube and the tube placed in a water bath at 40°. After 5 minutes 1 c.c. of 1 per cent. soluble starch solution is added and the mixture allowed to incubate for 30 minutes. At the end of this time 3 c.c. of saturated picric solution and 1 c.c.

[1] Myers and Killian, *Jour. Biol. Chem.*, 1917, xxix., 179.

of 20 per cent. sodium carbonate are added and the tube placed in boiling water for 15 to 20 minutes. It is then allowed to cool and diluted with distilled water in an accurately graduated cylinder until the intensity of the color approximates that of the standard (glucose in picric acid treated with sodium carbonate and heated), after which it is compared with the standard in the colorimeter. After correcting for the reducing power of the soluble starch, the activity is recorded in terms of the percentage of starch converted to reducing sugar.

Utilizing the principles outlined above it has been found possible to obtain a demonstrable amylolytic activity at a dilution of 1 to 400 when water was used as the diluent, and at 1 to 2,000 when dilution was made with 0.3 per cent. sodium chloride. For purposes of comparison a dilution of 1 to 100 (actually 1 to 200 allowing for the starch solution) appears to be the most suitable, with distilled water as the diluent. Although 0.3 per cent. sodium chloride is an excellent activator and represents the approximate content of sodium chloride in saliva, nevertheless with the handicap of a low chloride content, it appears possible to bring out greater individual variations than is possible otherwise. It may be noted that, with the dilutions employed, the variations do not appear to be influenced by the native content of sodium chloride in the saliva.

The method has been applied to a considerable number of normal individuals, the activity in the majority of cases falling between 30 and 45 when water was used as the diluent. With sodium chloride the variations were small, most of the figures falling between 46 and 50, suggesting the possibility that in some individuals a considerable part of the ferment is secreted in the zymogen form. Figures obtained on the same individuals at the same time of day agree very closely. The activity has been tested on representatives of a number of different nationalities and found to vary within essentially the same range as above. The same was true of a number of pathological cases including such conditions as diabetes, nephritis, gastric ulcer, etc. Several individuals were encountered, however, who for periods showed low activities, figures 10 to 20. Some of these had persistently suffered from gastric distress, others were suffering from acute in-

fections. An entirely satisfactory explanation for these low values, however, is not apparent. This is a topic to which we plan to give further study.

With the method it is possible to demonstrate a considerable decline in the activity of the saliva as a result of the glandular fatigue produced by the continuous secretion of saliva during paraffin chewing. Our results likewise indicate that the method is a very suitable one to employ in demonstrating the diurnal variation.

14 (1389)

## Parathyroids and calcium metabolism.

By **E. UHLENHUTH.**

[*From the Rockefeller Institute for Medical Research, New York City, N. Y.*]

Larvæ of the salamanders *Amblystoma maculatum*, *Amblystoma opacum* and *Eurycea bislineata* when fed on thymus exhibit severe tetanic convulsions. In contradiction to other amphibian larvæ which do not show this reaction, they possess no parathyroids; the tetanic convulsions of the thymus-fed salamanders are identical with a true parathyreoprival tetany.

The tetanic action of the thymus is due to the presence in the organ of the tetany toxin which can be antagonized only by the parathyroids, but not by the substances contained in normal food. The addition to the thymus diet of normal diet in amounts sufficiently large to keep a salamander larva in a perfectly normal condition does not prevent the tetanic convulsions. Tetany is caused by a toxic substance contained and excreted by the thymus.

Calcium salts, when dissolved in the water in which the thymus-fed larvæ live, do suppress, to some extent, the tetanic convulsions. Magnesium salts, however, assert the same action and are still more effective than calcium salts. The suppression of the tetanic convulsions by the salts of calcium is not a specific action of the calcium.

Neither calcium nor magnesium is able to prevent the development in the thymus-fed larvæ of severe and permanent lesions of the muscular system, lesions which are caused probably by

injuries inflicted upon the central nervous system by the tetany toxin. These nervous lesions develop even in the presence of calcium or magnesium and even without any convulsions at all, and finally lead to the death of the animals.

The most dangerous and important action of the tetany toxin is its highly injurious effect upon the central nervous system as expressed by the lesions of the muscular system (permanent spasmodic contractions and paralysis). The most important function of the parathyroids is to prevent the tetany toxin from coming in contact with the central nervous system, and this function cannot be substituted by any substance such as calcium or magnesium, since these substances do not antagonize the tetany toxin.

15 (1390)

**Malignancy of the crown gall and its analogy to animal cancer.**

By ISAAC LEVIN and MICHAEL LEVINE.

[*From the Department of Cancer Research of the Montefiore Hospital and Home, New York.*]

In a study reported recently on the influence of X-Rays on the development of the crown gall the writers have come to the conclusion that this growth presents an ideal material for the cellular study of the cancer problem. Dr. Erwin F. Smith, of Washington, considers this parasitic disease of plants to be identical with human cancer to such an extent that since crown gall is caused by a microörganism he maintains that all human cancers must be due to the same parasite. It seemed desirable to repeat Smith's experiments from the standpoint of human pathology and this was the object of the present investigation.

A large number and a great variety of plants were inoculated with a pure culture of *Bacterium tumefaciens* and a gross and microscopical study of the resulting crown galls was made. The analysis of the material shows that a certain number of these plant-tumors behave morphologically as well as biologically as benign growths. They grow very slowly, do not interfere with the development of the inoculated plant, compress but do not injure the neighboring normal tissues. Other crown galls appear to

be true malignant tumors. They dwarf the inoculated plant. The parts of the inoculated stem become necrotic above and even below the point of inoculation. Microscopically the galls show invasion and destruction of the neighboring normal tissues. In accordance with the findings of Smith a number of crown galls were obtained containing leafy shoots. Smith considers the latter condition to be analogous to human embryomata. A close microscopical study of the crown gall revealed characteristics which differ materially from the conditions obtained in animal cancer. In the majority of the specimens investigated the entire gall presents a uniform morphological appearance of small, young, undifferentiated cells. In other tumors the central growing part presents the usual appearance of a crown gall, while the periphery shows the development of adult differentiated tissue (parenchyma). This parenchyma is a part of the new growth and not of the normal tissues of the inoculated plant. The same is true of rudimentary organs (conducting system) or even a whole rudimentary organism (leafy shoot) which may appear at the periphery or in other parts of the ordinary crown gall. Such an appearance of highly differentiated tissues subsequently to and as a part of the development of a malignant tumor is unknown in animal cancer.

The conclusion to be arrived at from this study is that a fast developing simple crown gall presents a great deal of analogy to animal cancer and offers an ideal material for the cellular study of the latter condition. On the other hand the structure of the growing central part is identical in practically all the crown galls investigated thus far. It represents therefore only one type in the large group of pathological processes designated under the common name of cancer. It is hardly possible to assert on the basis of the study of the crown gall that all human cancers are formed through the activity of one and the same microörganism.

16 (1391)

## A method of preparing pure dihydrochloride of diaminodioxyarsenobenzene.

By PHILIP ADOLPH KOBER.

[*From the Division of Laboratories and Research, New York State Department of Health, Albany, N. Y.*]

The synthesis of a pure arsphenamine or salvarsan, in spite of the excellent published work of Ehrlich and Bertheim[1] and their collaborators, is still a vital problem. It is known by those who have given attention to the subject that the toxicity of arsphenamine varies and that batches from individual manufacturers vary more than they can account for in their procedures. Furthermore, as it seems fairly well proven that even Ehrlich's own manufacturers are unable to keep up a uniformly high standard,[2] it is evident there are some factors which are not understood or under control.

In studying this subject, I came to the idea that the toxicity of arsphenamine is largely due to the use of methyl alcohol and ether in the precipitation of the dihydrochloride and therefore made experiments to prepare the arsphenamine in aqueous solutions, free from any extraneous and objectionable substances:

Finding that the dihydrochloride of the salvarsan base was insoluble in excess of chlorides, as might be expected from the Law of Mass Action, an excess of hydrochloride acid was tried in salting out the drug. When first tried by making an aqueous solution of the dihydrochloride directly from the base, by dissolving in two normal sodium hydroxide and adding a slight excess of hydrochloride acid, and pouring the solution of hydrochloride into a strong solution of hydrochloric acid (1–1), a white precipitate was formed which, however, turned to a dark-colored gum. This transformation of the white precipitate into the black gum, as will be shown later, was due simply to coalescence of the particles. To prevent this coalescence three factors were changed: (1) The precipitation was conducted at a low temperature and (2) under more dilute conditions and (3) with vigorous stirring.

[1] *Ber.*, 45, 756, 1912.

[2] Roth, Hygienic Laboratory Bulletin, 113, p. 7, 1918.

Toxicological tests of the arsphenamine made by this method have been favorable but this study is not yet complete. While the physical properties are somewhat different than the usual arsphenamine, which has one molecule of methyl alcohol in its solid or crystal form according to Ehrlich and Bertheim, the chemical tests, analyses and toxicological reactions of the new substance are in harmony with pure dihydrochloride of diaminodioxyarsenobenzene with one or two molecules of water of crystallization.

Our experience with the methyl alcohol method has brought to our attention three possible objections against using this method:

(*a*) Our main objection to the methyl alcohol or any other similar solvent is based on the idea that it is easily oxidized or reduced, and as a concomitant with arsphenamine, a substance easily oxidized to very toxic and dangerous products, is apriori not a safe thing to have. In a subsequent paper we expect to prove these and other points.

(*b*) Our second objection to this method is expense. Even in peace times this method would be expensive.

(*c*) Our third objection is that these solvents are highly inflammable.

The advantages of the hydrochloric acid method are:

(*a*) The medium of precipitation, both the water and the hydrochloric acid can be absorbed by common and inexpensive absorbents and they are not easily oxidized or reduced.

(*b*) It is an inexpensive method, as the excess hydrochloric acid can be recovered ready for use by simple distillation.

(*c*) It is a non-inflammable method.

(*d*) It is pharmacologically more suitable and lèss open to question.

(*e*) The product seems more stable and less liable to oxidation, when exposed to the air.

(*f*) The method can be used for reprecipitation and is chemically better calculated to eliminate impurities as it is the same method used to obtain chemically pure sodium chloride.

17 (1392)

## On the antiseptic action of benzyl alcohol.

By D. I. MACHT and D. E. NELSON.

[*From the Pharmacological Laboratory of the Johns Hopkins University, Baltimore, Md.*]

In a communication[1] dealing with the pharmacological and therapeutic action of benzyl alcohol as a local anesthetic one of the authors (M.) called attention to the fact that pure benzyl alcohol when injected subcutaneously or intramuscularly was irritant and produced necrosis of tissue. In every case, however, in which this occurred there was never a pyogenic infection noted; the slough being of a sterile character. This, it was remarked, was undoubtedly due to the antiseptic properties of pure benzyl alcohol and the destructive effect was not at all surprising as similar results could be produced by antiseptics in general when injected into the tissues in the undiluted form. It was interesting, however, to investigate further the antiseptic properties of phenmethylol or benzyl alcohol, and especially in dilute form. In the present communication the authors wish to report a few observations on the subject.

Bacteriological studies with solutions of benzyl alcohol in water showed that it is quite antiseptic to a number of microorganisms. Experiments with a 0.5 per cent. solution of phenmethylol were found to kill cultures of Friedländer bacillus within nineteen hours. The same strength of the drug killed *pyocyaneus* cultures within twenty-four hours and growths of *bacillus coli communis* in seventy-two hours. Experiments with a 1 per cent. solution of benzyl alcohol gave evidence of even more marked and rapid bactericidal action.

A large number of clinical histories seem to confirm the authors' observations of the antiseptic properties of benzyl alcohol. A study of over 200 post-operative histories of patients on whom operations were performed with the use of benzyl alcohol as a local anesthetic showed that in all cases the wounds healed rapidly

[1] Macht, *Jour. of Pharmacol. & Exp. Therap.*, 1918, XI., 263.

and without any infection, such as was occasionally noted in cases in which ethyl chloride had been used. In all the above cases the concentrations of the benzyl alcohol employed ranged from 0.5 per cent. to 4 per cent. and such solutions were never found to be noticeably irritant to the tissues. As far as the authors have been able to learn none of the other commonly used local anesthetics such as cocaine, novocain, alypin, etc., can be said to possess antiseptic properties. It is therefore interesting to call attention to the antiseptic properties of benzyl alcohol as a desirable concomitant of its anesthetic action.

18 (1393)

## On the action of opium alkaloids on Trypanosoma brucei.

By **D. I. MACHT** and **J. WEINER.**

[*From the Pharmacological Laboratory of Johns Hopkins University and James Buchanan Brady Urological Institute, Baltimore, Md.*]

In a paper on the "Toxic Action of Opium Alkaloids Individually and in Combination with Each Other on Paramecia," by Macht and Fisher,[1] it was shown that some of the opium alkaloids were very toxic for that organism while others produced very little effect on it. It was found that the benzyl-isoquinolin group of alkaloids of which papaverin is the principal representative killed paramecia very quickly; whereas the pyridin-phenanthrene group of which morphin is the principal member was comparatively non-toxic. A further analysis of the papaverin action proved that the toxicity of that alkaloid was to be ascribed to the presence of the benzyl radicle in its molecule. Following the above investigation, it was interesting to inquire into whether the opium alkaloids are also toxic for other forms of protozoa, and especially for trypanosomes. The present authors have accordingly undertaken the study of the action of various opium alkaloids and their derivatives on *Trypanosoma brucei.* The organisms were obtained through the kindness of Dr. Wade Brown, of the Rockefeller Institute for Medical Research.

[1] Macht and Fisher, *Jour. of Pharmacol. and Exp. Therap.*, 1917, x., 95.

Experiments in vitro revealed as in the case of paramecia that the papaverin group of opium alkaloids was very toxic for trypanosomes. Papaverin itself in dilutions of 1 to 10,000 markedly inhibited the movements of the organisms in a few minutes and killed them completely within thirty minutes. Similarly the alkaloids narcotin and narcein were also found to be very toxic for trypanosomes in vitro.

Contrary to expectations however the morphin group of alkaloids while not as toxic as papaverin was also found to be deleterious to the trypanosomes, but in a much lesser degree. Dilutions of morphin sulphate 1 to 2,000 did not kill the organisms within an hour but were found to inhibit slightly their movements after the lapse of some ten minutes. Stronger solutions of morphin, for example 1 to 2,000, inhibited the movements and killed the parasites in less than half an hour. A similar action was noted in cases of codein and thebain. Heroin was found to be more toxic than morphin and killed the organisms in about fifteen minutes.

A comparative study of various alkaloids in addition to those already mentioned, namely, benzyl morphin or peronin, cotarnin, hydrastin, hydrastinin, and others seemed to point as in the case of paramecia to the benzyl grouping as the toxophoric portion of the papaverin molecule.

Having studied the effect of papaverin on trypanosomes in vitro some experiments were made on infected rats to ascertain whether that alkaloid could be employed in curing the infection with trypanosomes *in vivo*. It was found that small doses of papaverin had no effect in shortening the course of the disease when injected into rats. Neither were small doses of benzyl benzoate or benzyl alcohol efficient in this respect. These findings were not at all surprising as it is well known that experimental results with anti-syphilitic drugs in vitro can not at all be taken as an index to the clinical use of the same.

19 (1394)

## The spontaneous development of an acidosis condition in decerebrate cats.

By **J. J. R. MACLEOD.**

[*From the Physiological Department, Toronto University, Canada.*]

Investigations of the nature of the control of the respiratory center are rendered difficult because of the extreme susceptibility of the center to anesthetics. Much of the recent work has accordingly been done on man by methods suggested by Haldane and his pupils, and subsequently employed by Hasselbach, Linhard, R. G. Pearce and others. The obvious limitations to investigations of this type have prompted some investigators to employ decerebrate animals, or those in which the medullary centers are kept alive by artificial perfusion. The objections to the latter type of observation are too well known to require further comment here; they may or they may not be such as to render the results inapplicable to the intact animal. The chief objection to the use of decerebrate animals lies in the fact that the reactivity of the isolated centers is uncertain. This is particularly so in the case of the respiratory center. Some animals retain for several hours after the decerebration, a uniform and regular respiratory rate and volume, whilst others show an abnormal type of breathing. These irregularities, apparent in the work of Porter, Means and Newburgh, were also observed in the animals used by my former associate, R. W. Scott, in whose experiments it was further noted that apart from the animals that failed to breathe properly from the start, there were others which were apparently perfectly normal in this regard for some time (1–2 hrs.) after the decerebration, but in which later the breathing became dyspneic and irregular, and death soon followed, usually after an acute attack of vomiting.

As a preliminary to an investigation into the nature of the respiratory hormone, it was considered essential to investigate the cause of this delayed dyspnoea of decerebrate animals, not alone because these are probably the most suitable for use in such investigations, but also because the behavior of the abnormal an-

imal strongly suggests the possibility that development of a condition of acidosis is responsible for the symptoms. Some of the most conspicuous of the results so far obtained are reported here.

CAT. No. XXII.

| Time after Decerebration. (Min.). | Respiration per Min. | | Alv.-$CO_2$ (Per Cent.). | Blood-$CO_2$ (Per Cent.). | Blood $P_H$. | Blood L.A. (Per Cent.). | Urine. | | Rect. Temp. °C. |
|---|---|---|---|---|---|---|---|---|---|
| | c.c. | Rate. | | | | | $\frac{N}{10}$ Acid (Per Cent.). | $NH_3$ Per Cent.). | |
| 53 | 1125 | | | | | | | | |
| 70 | | | 3.5 | | | | | | |
| 73 | | | 3.6 | | | | | | |
| 78 | 1080 | 28 | | | | | | | 39 |
| 93 | | | | | | | 30 | | |
| 108 | 1225 | 28 | | | | | | | |
| 118[1] | | | 3.3 | | | | | | 40 |
| 133 | | 38 | | | | | | | |
| 138 | | | 2.9 | | | | | | |
| 148 | | | | | 7.4 | | | | |
| 161 | | 38 | | | | | | | |
| 171 | | | 3.0 | | | | | | 40 |
| 178 | | 44 | 1.6–1.8 | | | | | | |
| 203[2] | | | 1.7 | | | | | | |
| 208 | | | | 24.4 | 7.1 | 0.296 | 30 | | |

CAT. No. XXIII.

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | 0.107 | |
| 90 | | | | | | | 106 | 0.076 | 38.5 |
| 135 | 1080 | 27 | | | | | | | |
| 140 | | | 3.3 | | | | | | |
| 170 | | | | | | | | | |
| 195[3] | 1120 | 28 | | | | | | | |
| 210 | 1170 | 28 | | | | | | | |
| 215 | | | 3.3 | | | | | | |
| 230 | 1150 | 30 | | | | | 20 | 0.326 | |
| 250 | 1120 | 28 | | | | | | | |
| 255 | | | 2.8–3.0 | | | | | | |
| 285 | | | 2.9 | | | | | | |
| 290 | 950 | 26 | | | | | | | |
| 293 | | | 2.9 | | | | | | |
| 295 | 930 | 24 | | | | | | | |
| 302 | | | | | 7.6–7.7 | | | | |
| 304 | | | | 45.0 | | 0.098 | 6.5 | | |
| 305 | 960 | 22 | | | | 0.101 | | | |

The animals (cats) were decerebrated by the method of Miller and Sherrington. In those on which regular breathing returned, an interval of one hour was allowed to elapse, so that the

[1] Suddenly hyperpnœic.

[2] Vomited.

[3] Rigidity slight.

influence of the anesthetic (ether) might have ample time to disappear, and then observations were made on the following: (1) The minute volume of air breathed; (2) the alveolar $CO_2$; (3) the total $CO_2$, $P_H$ and (5) the lactic acid content of the arterial blood; and lastly, (6) the total acid excretion by the urine.

The general nature of the results is indicated in the following table in which the above values are given for an animal which showed no dyspnea (XXIII), and one in which this and irregular breathing were pronounced (XXII).

These experiments typify the results in extreme cases; the animal in XXIII remained in perfect condition for over five hours after the decerebration, whereas in that of XXII the breathing, although normal at the start, became later rapid and dyspneic, death, preceded by vomiting, occurring in about three and one half hours after the decerebration. Of a total of thirteen animals so far observed, six behaved like XXIII, for at least five hours, and four like XXII, while three gave intermediate results. Animals in both of the latter groups died within three hours. In the animals of the second group which provisionally we may call the acidosis group, the following changes were invariably found: (1) A progressive decrease in alveolar $CO_2$ followed later by (2) a decrease in blood $CO_2$, (3) an increase in acidity ($P_H$ lower and (4) an increase in the lactic acid content of the blood. The excretion of acids and ammonia by the urine was irregular. The simplest interpretation of the results is that the development of a condition of acidosis is responsible for the changes observed in the dyspneic group of animals. It is further of interest to record, that decerebrate rigidity was much more pronounced in the "acidosis" animals than in those that remained normal. Whether the rigidity is responsible for the acidosis, by causing lactic acid to be discharged in excessive quantities into the blood, or whether it is an effect of the acidosis, is at present problematical.

Marked glycosuria was common in most of the animals.

20 (1395)

## Study of the chemistry of pernicious anemia.

By JOSEPH BARSKY and MAX KAHN.

[*From the Department of Laboratories, Beth Israel Hospital, New York City.*]

In the study of three cases of pernicions anemia we found a condition of marked intestinal putrefaction, with a reduction of the detoxication functional capacity of the liver as evidenced by the sulfo-conjugation test. While the functional activity of the pancreas is normal, the intestinal digestion, investigated by means of the Schmidt-Strassburger test diet, shows failure to absorb the normal amount of nitrogen, there being a very high nitrogen loss through the feces; the bulk of the feces is increased; the fat elimination is normal. The kidney function is normal. A study of the functional activity of the stomach by means of the fractional method reveals a complete anacidity, a total absence of pepsin, an increased gastric residuum, and a negative gastroalbumorrhea test. Examination of the duodenal contents, following the investigations of Schneider,[1] shows that the excessive hemolysis of pernicious anemia is attended by both a pleochromie and a urobilinocholie. The patients showed evidence of acidosis, as shown by the carbon dioxide of the plasma and of the alveolor air and by the H′ ion concentration of the blood. The nitrogen partition of the blood is normal, except for the high creatinine figure. The blood showed an increased ash and lime content, and a normal glucose, cholesterol and total lipin percentage. The urinary nitrogen partition was normal. The urinary oxyproteic acid fraction was increased. There was a marked increase in the ethereal sulfate and neutral sulfur fractions of the urine.

[1] Schneider, J. P., *Arch. Int. Med.*, 1916, XVII, p. 32.

# SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

**Ninety-fifth meeting.**

*Rockefeller Institute for Medical Research, December 18, 1918.*
*President Gies in the chair.*

21 (1396)

## The effect of organic acids and their amido-compounds on the hydration of agar and on a biocolloid.

By **D. T. MACDOUGAL** and **H. A. SPOEHR.**

[*From the Desert Laboratory, Tucson, Arizona.*]

Earlier researches on hydration effects were concerned chiefly with gelatine as taken to represent the action of living matter. The effort to obtain results directly applicable to plant protoplasm, led to a consideration of chemical composition, solubility and other properties as a consequence of which mixtures of agar and protein were found to give hydration reactions so nearly comparable to those of living cell-masses that their study yields extremely useful facts and numerous stimulating suggestions.

The importance of the acids or of the hydrogen ion concentration in swelling had become apparent very early in the brief history of this subject. Workers using botanical material have dealt with this matter chiefly by the use of hydrochloric, and with acetic, citric and malic acids, and with salts of biological occurrence and concentrations. Although much careful work has been done some doubt still exists as to the part which the acid radicle may play in the complex system of substances always present in the protoplasmic colloid. A study of the action of these acids and of organic acids and of their amido compounds promised comparisons that might be of importance in this matter, and a series of measurements were carried out in the equable temperature chambers of the Coastal Laboratory August to November, 1918.

Three groups of substances were selected for these tests; (1) succinic acid and its amido-compound, *a* amino-succinic or aspartic, which are dibasic, and its amide, asparagin, which is monobasic; (2) acetic acid and *a* amino-acetic acid or glycocoll, which are monobasic; (3) propionic acid and *a* amino-propionic acid or alanin, also monobasic.

The swellings of sections of agar and a mixture of 8 parts agar and 2 parts oat-protein are selected for discussion at the present time. The measurements were made with the type of auxograph which has been previously described. The principal end results are given in the following table.

Hydration of agar and agar-oat-protein in organic acids and their amido-compounds at 16°–17° C. Trios of sections .10–.23 mm. in thickness from plates cast from 2.5 per cent. solutions were used. Such plates were shrunken in thickness by dehydration in 48–60 hours at 18°–22° C. Duration of swelling 20–60 hours. Expansion in percentages of dried thickness.

| Concentration. | Water Dis. | Succinic Acid Mol. | Aspartic Acid Mol. | Asparagin Mol. | Acetic Acid Normal. | Glycocoll Mol. | Propionic Acid Normal. | Alanin Mol. |
|---|---|---|---|---|---|---|---|---|
| | | | | *Agar.* | | | | |
| | 2413% 2739 | | | | | | | |
| 0.05 | | 1091 | 827 | 2308 | 1433 | | 1200 | 1474 |
| 0.01 | | 1273 | 1270 | 2365 | 1560 | 2965 | 1300 | 2421 |
| 0.002 | | 1600 | 1400 | 2440 | 1790 | 3166 | 1625 | 2790 |
| 0.0004 | | 1750 | 1788 | 2720 | 1955 | 2605 | 1800 | |
| 0.00008 | | 2528 | 2080 | 3250 | 2640 | | | |
| | | | | *Agar* 8, *Oat-Protein* 2 *Parts* | | | | |
| | 2100% 2630 | | | | | | | |
| 0.05 | | 700 | 855 | 1867 | 1090 | 1938 | 800 | 2046 |
| 0.01 | | 864 | 900 | 2455 | 1255 | 2340 | 1000 | 2317 |
| 0.002 | | 909 | 1670 | 2523 | 1738 | 3050 | 1250 | 2410 |
| 0.0004 | | 1136 | 2600 | 2675 | 2238 | 3000 | 1591 | 2273 |
| 0.00008 | | 2330 | 3050 | 2600 | 2480 | | 1864 | |

The deductions to be drawn from these figures, all being averages of 3 to 9 measurements, are numerous, but attention must be confined to a few pertinent cases.

1. It is to be seen that equimolecular concentrations of the three organic acids present small divergence of effect on agar and more positive differences in agar-protein.

2. Agar swells more in succinic acid than in its amido-com-

pound but reverses this relation notably in the acetic-glycocoll couple and in the propionic-alanin pair.

3. The agar-protein biocolloid shows notably greater hydration in the amino-acids than in the related organic acids, and greater even than the hydration in distilled water.

4. Equimolecular concentrations of amino-acids produce notably greater swellings of the biocolloids in comparison with related organic acids implying the positive action of factors other than the hydrogen ion concentration.

5. Glycocoll facilitates hydration in all concentrations above .01 M. in both agar and agar-proteins, and also in agar-gelatine, the data of which are not given in this paper. This fact goes far in explanation of the scattered results obtained by various workers in which accelerated growth or increased total growth has been seen to result from the addition of glycocoll to nutrient solutions. Such increases have been attributed to catalytic action by Dakin and others.

6. The amide, asparagin, induces a maximum hydration, greater even than that possible in agar in distilled water, and very high at all concentrations. Similar action was exerted on agar-gelatine and agar-protein plates. The positive action of both glycocoll and asparagin is indicated by the fact that the maximum effect is reached at certain concentrations above the minimum.

The destruction of our supply of phenyl-alanin in transit prevented an examination of the effects of this substance, but it will be possible to extend this work to this and other amides in the next few months.

22 (1397)

## The carbon dioxide of injury and of respiration in nervous tissue.

By **A. R. MOORE.**

[*From the Physiological Laboratory of Rutgers College, New Brunswick, N. J.*]

It has been shown that injured nervous tissue gives an acid reaction with phenolsulphonephthalein as indicator and that the acid measured by Haas[1] method is carbon dioxide.[2] If the rate

[1] Haas, A. R. C., *Science*, N.S., vol. 44, pp. 105–108, 1916.

[2] Moore, A. R., PROC. SOC. EXP. BIOL. AND MED., vol. 15, pp. 18–19, 1917.

of acid output of a frog's sciatic nerve be measured by taking the time required for the solution to change tint from pH 7.8 to pH 7.4, this rate will be seen to fall after the first or second consecutive readings. If now the nerve be taken out and crushed with a glass rod, the subsequent measurement will show a large increase in

FIG. 1.

rate, which, in turn, after a few readings, falls to the former low rate. The following determinations on a pair of frog sciatic nerves shows this fact.

| | | Time. | | Rate. |
|---|---|---|---|---|
| Fresh, | first | reading | 360 seconds | .28 |
| " | second | " | 1440 " | .07 |
| Crushed | first | " | 420 " | .24 |
| " | second | " | 1740 " | .06 |

$$\text{Rate} = \frac{100}{\text{Time}}$$

It is apparent that the acid resulting from injury develops only momentarily and is soon washed out by the surrounding neutral Ringer's solution. It therefore seems reasonable to suppose that the carbon dioxide output during the time following is largely that of respiration.

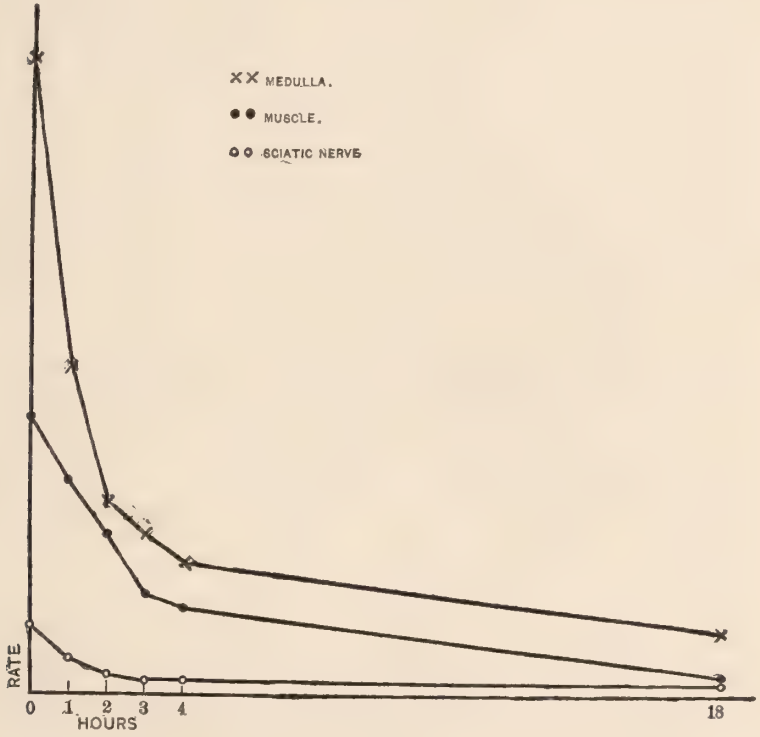



FIG. 2.

With a view to comparing the rates of acid production of central nervous system, muscle and nerve fiber, equal weights of the medulla, the sartorius muscle, and the sciatic nerves of the

same individual frog were taken. The Haas measurements were made by putting the tissue to be tested into a small test-tube containing 3 c.c. of Ringer's solution tinted with the indicator and previously rendered pH 7.8 with a drop or two of $n/10$ NaOH to the liter. The tube was closed with a paraffined cork. A bubble of air acted as a stirrer and the preparation was inverted and righted at short intervals until the tint of the solution matched that of a standard tube of solution whose pH = 7.4. Time was taken with a stop watch and the number of seconds required for this amount of change gives an inverse measure of the rate.

$$\text{Rate} = \frac{100}{\text{time in seconds}}.$$

As soon as one reading was taken the tube was emptied of its fluid and refilled from the stock bottle, and another reading taken. As a rule five or six consecutive readings were made. The last two or three were fairly constant; hence their average values are given in the tables and form the basis of the graphs. A set of readings was made each hour and in the interval the tissues remained in their respective tubes each containing 3 c.c. of solution (Table I., Fig. 1) or in beakers each containing 100 c.c. of neutral

TABLE I.

| Hour. | Medulla. | | Muscle. | | Sciatic. | | Sciatic / Medulla. | Sciatic / Muscle. |
|---|---|---|---|---|---|---|---|---|
| | Time. | Rate. | Time. | Rate. | Time. | Rate. | | |
| 0 | 18 | 5.5 | 20 | 5.0 | 110 | .91 | .16 | .18 |
| 1 | 45 | 2.2 | 55 | 1.8 | 180 | .55 | .25 | .30 |
| 2 | 80 | 1.25 | 120 | .83 | 360 | .28 | .22 | .34 |
| 3 | 85 | 1.17 | 150 | .67 | 900 | .11 | .09 | .16 |
| 4 | 60 | 1.7 | 40 | 2.5 | 300 | .33 | Av. = .18 | .245 |
| 5 | 60 | 1.7 | 60 | 1.7 | 400 | .25 | | |
| 6 | 90 | 1.1 | 120 | .83 | 600 | .17 | | |

TABLE II.

| Hour. | Medulla. | | Muscle. | | Sciatic. | | Sciatic / Medulla. | Sciatic / Muscle. |
|---|---|---|---|---|---|---|---|---|
| | Time. | Rate. | Time. | Rate. | Time. | Rate. | | |
| 0 | 30 | 3.3 | 70 | 1.43 | 270 | .37 | .11 | .26 |
| 1 | 60 | 1.7 | 90 | 1.11 | 540 | .185 | .11 | .17 |
| 2 | 100 | 1.0 | 120 | .83 | 1000 | .1 | .10 | .12 |
| 3 | 120 | .83 | 190 | .52 | 1200 | .07 | .08 | .13 |
| 4 | 145 | .69 | 220 | .45 | 1200 | .07 | .10 | .15 |
| 18 | 300 | .33 | 1000 | .10 | 1620 | .06 | .18 | (.60) |
| | | | | | | | Av. = .11 | .17 |

Ringer's solution (Table II., Fig. 2). In the small quantities of fluid a strong acid reaction developed during the hour. This led to rigor and opacity of the muscle by the end of the fourth hour. This change was accompanied by a marked increase in the rate of acid output not only in the muscle but in the medulla and in the sciatic nerves. Probably death changes were also occurring in the nervous tissues. Where sufficient solution was present to maintain neutrality, no secondary maximum of acid production developed.

It will be noted from the graphs and tables that a marked fall in rate occurs during the first hour, and that the medulla has a higher rate of acid production than muscle while the sciatic nerve has an extremely low rate. Under appropriately headed columns of the tables are given the ratios of rate of acid output in the sciatic nerves to that of the medulla and to that of the muscle of the same animal. In view of the fact that Mathews[1] states that the respiratory rate of nerve fibers is "higher than that of any other tissue examined," it is interesting to note that under identical conditions of experiment, nerve fibres produce carbon-dioxide at 10–20 per cent. of the rate of the medulla and at 15–30 per cent. of the rate of muscle.

23 (1398)

## Volumetric analysis of ion-protein compounds.

By JACQUES LOEB.

[*From the Laboratories of the Rockefeller Institute for Medical Research, New York.*]

The speaker demonstrates that gelatin at pH > 4.7 combines only with cations and at pH < 4.7 only with anions, while at the isoelectric point (pH = 4.7) it combines with neither anion nor cation.

He shows further that the curves representing the influence of monovalent anions or cations upon the swelling, osmotic pressure and viscosity of gelatin are always approximately parallel with the curves representing the amount of anion or cation found in chemical combination with the gelatin.

[1] Mathews, A. P., "Physiological Chemistry, p. 590, 1915.

24 (1399)

## Experimental pneumonia produced by Streptococcus hemolyticus.

By MARTHA WOLLSTEIN and S. J. MELTZER.

[*From the Laboratories of the Rockefeller Institute for Medical Research, New York.*]

Pulmonary lesions were produced by the insufflation of a strain of *Streptococcus hemolyticus* isolated in Texas and kindly given us by Dr. Avery. The cultures proved to be highly virulent for white mice. A quantity of the culture was insufflated intrabronchially into twenty-four dogs. The dose varied from 1 to 3 c.c. per kilo. Of these animals nine died in less than twenty-four hours, one died on the third day, and the rest were killed three to fifteen days after the inoculation. Dogs which survived more than two days went on to recovery, although they were ill for several days. Streptococcemia was found in all the animals which died early. It was also found in one of two dogs which was killed on the third day and in one of two killed on the fourth day after injection. Later than the fourth day no streptococci were found in the blood. Blood stained pus was present in the pleural cavity in three dogs, all having streptococci in the heart's blood. One of these animals died on the third day, one was killed on the third day and one on the fourth day. In the rest the pleura was normal. Empyema seemed to have developed before the third day.

The pneumonic lesion, in its early stage (twenty-four hours after injection), consisted of intense congestion, edema and small hemorrhages without pleurisy. After forty-eight hours the congestion and edema were still more marked and areas of bronchopneumonia had developed. The solid areas coalesced to some extent, but never became massive. The lungs in these dogs, even on the third day, were never very solid. The lesions involved usually more than one lobe. Resolution had begun on the fourth day, but in one instance there was a distinctly solid area of bronchopneumonia present on the seventh day. In the second week only areas of darker color were left. In no case had organization occurred.

Cultures from the lungs gave streptococci on the first, second, third and fourth days, but remained sterile on the fifth and sixth

days. One case which presented an area of bronchopneumonia still unresolved on the seventh day gave a growth of streptococci from that area. Later than the seventh day the lungs contained no streptococci.

Microscopic examination of the sections made from lungs within twenty-four hours after insufflation of the culture showed congestion of all the vessels with the formation of thrombi in some of them. The alveolar contents consisted of red cells and coagulated serum, but there were practically no hemorrhages. On the second day the microscope showed that the alveoli were packed with polynuclear cells, little fibrin and many red blood cells. The solid areas surrounded inflamed bronchi. Infiltration of the framework of the lungs was present but not intense in any case. An abscess had formed in one lobe in one of the three cases with empyema.

The pulmonary lesion produced by the insufflation of the *Streptococcus hemolyticus* resembled the lesion found in human lungs from which the same organism was cultivated in that it was a bronchopneumonia with marked edema and a large amount of hemorrhage; it differed however from the human lesion by the lack of any tendency toward organization. In the experimental series empyema occurred in 12 per cent. of the cases and a pulmonary abscess was present only once.

25 (1400)

## The prognostic value of the creatinine of the blood in nephritis.

By VICTOR C. MYERS and JOHN A. KILLIAN.

[*From the Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital, New York.*]

At the May, 1914, meeting[1] attention was called to the accumulation of creatinine in the blood in advanced chronic interstitial nephritis, data being reported on two cases at that time. It was then suggested that the retention of creatinine might be of etiological importance in uremia on account of its containing the toxic guanidine group, and further that the creatinine might be of considerable prognostic value in advanced nephritis. Further study

[1] Myers and Fine, PROC. SOC. EXP. BIOL. AND MED., May 20, 1914, xi., p. 132.

showed that as the permeability of the kidneys is lowered in conditions of renal insufficiency this becomes evident in the blood; first, by a retention of uric acid, later, by that of urea, and lastly by that of creatinine, indicating that creatinine is the most readily eliminated of these three nitrogenous waste products.[1] Theoretically, the amount of the increase of the creatinine in the blood should be a safer index of the decrease in the permeability of the kidneys than the urea, for the reason that creatinine on a meat free diet is entirely endogenous in origin and its formation (and elimination normally) very constant. Apparently the kidneys are never able to overcome the handicap of a high creatinine accumulation, for, we soon found that those cases in which the creatinine had risen above 5 mg. per 100 c.c. of blood rarely showed any marked improvement and almost invariably died within a comparatively limited time.[2] On the other hand, cases with high figures for urea, but without marked creatinine retention, generally showed improvement.

We have now had the opportunity of following 94 cases with creatinine values of 5 mg. or more. The outcome has been; died 83, unknown 3, unchanged 4, improved 2 and recovered 2. The two cases classified as recovered were acute cases in which the creatinine remained over 5 mg. for only a few days. Of the 83 known dead, 80 per cent. died in less than two months, although a few cases have lived as long as a year. There were a good many cases who were able to be up and about, and some who showed considerable clinical improvement. The creatinine gave us a better prognostic insight into these cases than either the blood urea or phthalein tests which were made simultaneously. It is our opinion that in these advanced cases of nephritis the blood creatinine furnishes a more reliable prognosis than any other test we possess.

[1] Myers, Fine and Lough, *Arch. Int. Med.*, 1916, xvii., p. 570.

[2] Myers and Lough, *Arch. Int. Med.*, 1915, xvi., p. 536.

26 (1401)

## Chemistry of the blood in scurvy.

By ALFRED F. HESS and JOHN A. KILLIAN.

[*From the Bureau of Laboratories, Department of Health, New York City.*]

The urea content, $CO_2$ combining power, percentage of sugar, diastatic activity and calcium content of the blood were ascertained in a number of cases of infantile scurvy. Two abnormal variations were found: (*a*) a moderate acidosis, figures under 40 or 45 obtained according to the Van Slyke method; (*b*) a deficiency of calcium. Neither appeared to be a basic factor or to run a course parallel to the scorbutic process.

| | Date. | Ca, (Mg. per 100 C.c) | Notes. |
|---|---|---|---|
| 1 | May 23 | 3.04 | No tetany. |
| 2 | " 14 | 6.9 | |
| | " 23 | 6.8 | Marked rickets. |
| 3 | " 22 | 7.2 | |
| | June 4 | 11.4 | Prune Juice (15 c.c.) for 12 days. |
| 4 | May 22 | 5.5 | Cod Liver Oil for 13 months. |
| 5 | " 22 | 3.2 | |
| | June 4 | 8.4 | Banana for 12 days. |
| 6 | " 22 | 4.5 | Lactose 8 days. |
| | " 4 | 11.0 | " 20 days. |
| 7 | Nov. 19 | 5.2 | Moderate rickets. |
| | " 26 | 5.6 | |

The accompanying chart shows the results of calcium tests carried out according to the Halverson and Bergeim deproteinization method with 5 c.c. or more of plasma. The normal content is about 10 mg., so that it will be seen that there was a striking calcium deficiency. None of the cases had convulsions; nor can the results be accounted for by the presence of tetany.

27 (1402)

## Carbohydrate fermentation by bacteria as influenced by the composition of the medium.

By J. BRONFENBRENNER and M. J. SCHLESINGER.

[*From the Department of Preventive Medicine and Hygiene, Harvard Medical School, Boston, Mass.*]

The value of the fermentation test among the methods at our disposal for identification of bacteria is generally accepted. And yet, every bacteriologist must have encountered in his experience a number of instances of apparently inexplicable inconsistencies in the results obtained by this test. Not only does it often happen that a given strain producing a large amount of acid or gas will occasionally produce very little, but at times indeed it produces none at all. In fact, the amount of gas produced by a bacterium at different times varies so widely, that at present it is suggested by some bacteriologists that the amount of gas produced by a given culture has no diagnostic significance. This point of view owes its existence merely to the fact that the amount of gas produced by a given culture has no diagnostic significance. This point of view owes its existence merely to the fact that the amount of gas produced by bacteria depends on too many factors to attempt to control them. In our work we came across inconsistencies in the amount of acid and gas production, but discovered that these inconsistencies were very often due to variations in the composition of the media. The study is indeed not finished, but even in its present stage it is quite convincing. Omitting the details of the experiment, which will be published in full later, we shall state here merely the general plan and the results obtained.

The experiment consisted in growing a strain of *B. coli*, which, in the original culture, produced very little acid or gas, upon a medium consisting of peptone-phosphate-lactose-water with the addition of an indicator permitting direct reading of hydrogen ion concentration developed in the growing culture.[1] The composition of this medium was varied in every possible direction.

[1] This indicator, consisting of China blue and rosolic acid, was described by us in the September issue of the *Journal of Medical Research*.

Thus, in all, there were 294 combinations, including variable amounts of peptone, lactose and phosphate from zero to and slightly above that used in ordinary media. Both the hydrogen ion concentration and the amount of gas produced were recorded daily for three weeks, thus giving us 294 curves.[1]

The analysis of these curves brought out the following facts:

Increase in the concentration of lactose generally increased the rate of acid production (given constant concentration of peptone and buffer-salts).

Thus, when the amount of buffer-salts is not sufficient, the increase in concentration of the carbohydrate inhibits the growth.

With any concentration of carbohydrate, the amount of free acid indicated depends of course on the concentration of the buffer-salts.

Concentration of peptone affects the amount of free acid indicated partly in the same manner as does the concentration of neutral salts, but in addition the amount of sugar attacked is smaller as the concentration of peptone in the medium is increased. Thus, when 1 per cent. or more of peptone is used in the medium, the amount of carbohydrate has to be increased in proportion in order to obtain sufficient amount of free acid (amount of neutral salts being constant).

The amount of gas produced changes inversely with variations in the hydrogen ion concentration, other conditions being equal.

Therefore, other conditions being equal, the amount of gas produced increases directly with concentration of buffer.

Given constant concentration of carbohydrate and buffer salts, the amount of gas produced changes directly with the concentration of peptone.

These findings suggest that the number of discrepancies in results of the fermentation experiments are largely due to the fact that media are not usually compared with sufficient attention to factors above mentioned. The variations in sugar content and especially in buffer content of the media are not guarded against sufficiently in routine procedure of making media. Moreover,

[1] While in this paper we refer only to the part of the experiment which dealt with the production of acid and gas, the complete records will cover also the data on nitrogen and carbohydrate metabolism, including the gas analysis and the curves of multiplication.

it has been the custom to have the same concentration of the different ingredients in carbohydrate media. The usefulness of media could be greatly increased if the composition of each medium were more closely adapted to the purpose in hand. Thus, for isolating bacteria on a plate by the process of excluding all the lactose-fermenting bacteria, one should have the initial reaction as nearly neutral as possible; one should incorporate as high an amount of lactose as consistant with the concentration of peptone in the medium; one should buffer the medium only enough to prevent excessive penetration of the acid into the surrounding agar, but not so much as to delay the appearance of free acid within and immediately around the colony. On the other hand, when preparing a liquid medium in which one desires to follow gas formation in addition to that of acid, one should be careful to have a fair excess of buffer. Even a small amount of free acid produced can be easily distinguished in a comparatively thick layer of a tube of liquid culture; thus the excess of buffer will not interfere materially in diagnosing the acid production, but it will greatly improve the growth by keeping down the hydrogen ion concentration and will facilitate the production of gas. Again, if one desires to follow the growth of bacteria through a longer period of time in order to determine the character of later stages of metabolism (late alkalinity for instance) one should correlate the respective amounts of carbohydrate and peptone. If one desires to determine the power of reduction, one must use a different indicator from that which ought to be used if the free acidity alone is to be determined, for in that case one should select an indicator which is not easily affected by the reduction.[1]

[1] This work is a part of the investigation of food poisoning, conducted under the direction of Dr. M. J. Rosenau, professor of preventive medicine and hygiene, Harvard Medical School. The investigations are done under the auspices of the Advisory Committee on the Toxicity of Preserved Foods of the National Research Council, and under a grant of the National Canners Association.

# SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

**Ninety-Sixth meeting.**

*College of Physicians and Surgeons, January 15, 1919.*
*President Gies in the chair.*

28 (1403)

## The mechanism of corpuscle and serum anaphylaxis in the rabbit.

By ARTHUR F. COCA.

[*From the New York Orthopedic Hospital, New York.*]

1. In rabbits dying acutely after a reinjection of corpuscles or after a primary injection of pig's corpuscles, the pulmonary circulation is found to be impermeable to saline solution under pressures so much greater than the maximal normal blood pressure in the pulmonary artery of that animal (over four times as great in some instances) that a sufficient immediate cause of death under these circumstances is seen in this physiologically complete interruption of the pulmonary circulation.

2. This obstruction of the pulmonary circulation is not due to an agglutination of the corpuscles and it may reasonably be referred to an effect on the muscular coat of the arterioles because the same phenomenon is produced by the injection of dissolved corpuscles and by the reaction of acute serum anaphylaxis—active and passive—and also by the introduction of the antigen into the pulmonary circulation after the latter has been perfused for five minutes with saline solution.

3. In the light of these observations, the local effect of Arthus would seem reasonably explicable as an area of infarction due to the interference with the blood supply to the area.

4. The cachexia and late death of rabbits in serum anaphylaxis offered, in the one instance of this kind, examined, a pathological

picture consonant with the findings in acute anaphylaxis in the rabbit—the usual picture of a chronic interference with the circulation; namely, anasarca and greatly dilated right heart.

5. In the guinea-pig dying in acute anaphylaxis, the pulmonary circulation offers no increased resistance to the passage of fluid through it.

29 (1404)

## The effect of oxidation on Wassermann antigen.

By REUBEN OTTENBERG and ARTHUR KNUDSON.

[*From the Department of Bacteriology, and of Biological Chemistry, Columbia University, New York.*]

In 1914 the authors had occasion to prepare some lecithin by MacLean's method (*Journal of Pathology and Bacteriology*, 18, p. 490). The method (an elaborate one) consists essentially of numerous precipitations out of ether and water, by means of acetone, of the alcoholic extracts of dried beef heart. The purified precipitate is finally dried in vacuo over sulphuric acid. Throughout the work air is excluded so far as possible.

In the present work from 6½ kilos of lean pressed beef heart, about 8 grams of purified lecithin were obtained. The lecithin had a yellowish white waxy appearance and upon analysis was found to contain 4.06 per cent. phosphorus and 1.93 per cent. nitrogen. The nitrogen and phosphorus are in the ratio of one to one. It had an iodine number of seventy. Throughout the procedure every precaution was taken to exclude air by replacement with $CO_2$ so as to prevent oxidation as far as possible. Some of the lecithin was put in tubes in vacuo, and some in carbon dioxide gas.

The lecithin separated in this way is not true lecithin, but a mixture of true lecithin and kephalin. It possesses however all the properties of the substance generally alluded to as lecithin.

The samples of lecithin put up in tubes have been tested in the Wassermann reaction at various times during the last four years. It has been found that the antigenic property of those tubes which remained perfectly sealed was preserved and remained

of practically the same titer as it was at the beginning. On the other hand, a considerable number of the tubes developed small cracks which admitted some air. The dried antigen in these tubes changed in appearance, becoming very dark and confluent. This presumably oxidized lipoid was found to have lost its antigenic value entirely and also to have developed considerably more anticomplementary property than the original had.

The properties of the original preparation as an antigen were about equal to those of the usual lecithin extracted from beef heart. Complete fixation was obtained at a dilution of about 1–180,000 of the lipoid. Slight anticomplementary effect was evident at a dilution of 1–7,500. This gives a ratio between antigenic and anticomplementary doses of about 1–25.

MacLean's reason for devising this method of obtaining a pure lecithin was to avoid the oxidation of the unsaturated fatty acid radical in the lecithin. From our observation the same process, oxidation, is what ordinarily results in the loss of antigenic value; antigen can be preserved indefinitely under anaërobic conditions. It is possible that this indicates that the antigenic value is dependent on the presence of the unsaturated fatty acid radical (oleic acid).

30 (1405)

## Changes in the concentration of the carbon dioxide of the blood following changes in the circulation through the medulla oblongata.

By F. H. PIKE, HELEN C. COOMBS and A. BAIRD HASTINGS.

[*From the Department of Physiology, Columbia University, N. Y.*]

It is desirable and even necessary, in attempting to estimate the rôle of the afferent nerves in the regulation of the respiratory movements[1] to investigate more carefully the effects upon respiratory movements of changes in the volume of blood flowing through the medulla oblongata.[2]

[1] Pike, F. H., and Coombs, Helen C., *American Journal of Physiology*, 1918, vol. 45, p. 569; this journal, 1918, vol. xv., p. 55.

[2] Pike, F. H., *Science*, 1918, xlvii., pp. 121–122.

The experiments described here were done on cats. Etherization and tracheotomy were routine procedures. Some of the cats were decerebrated, after which no more ether was necessary. The two common carotid and two vertebral arteries were isolated in the neck so that they might be ligated temporarily or permanently. Blood pressure was taken from the left carotid artery and the respiratory movements recorded by a Crile stethograph and a Verdin tambour. Blood for the estimation of the carbon dioxide was drawn from the femoral artery, and the estimations were made by the Van Slyke method. Changes in the volume of blood flowing through the medulla were brought about by occluding the cerebral arteries. Control samples of blood were taken at the beginning of each experiment. Other samples were taken during the period of occlusion of the cerebral arteries and after the release of these arteries and the restoration of the cerebral circulation. In some of the experiments, the arteries were never occluded, but changes in the blood flow to the medulla were produced by successive small hemorrhages of eight to ten cubic centimeters each. Pulse counts in some of these experiments were made by Miss Ethel Wickwire.

Simple occlusion of the cerebral arteries was sufficient to produce severe dyspnœa in some animals, but in others, dyspnœa appeared only after a considerable quantity of blood had been drawn. In all animals, the effects of the occlusion became more and more severe as more and more samples of blood were drawn. Hemorrhage alone, if carried sufficiently far, is followed by dyspnœa. Any injury to the spinal cord which prevents the compensatory rise of systemic blood pressure is followed by more severe dyspnœa on occlusion of the cerebral arteries than otherwise results.

The changes in the total carbon dioxide of the blood attending occlusion of the cerebral arteries are shown in the following table.

It is seen that the total carbon dioxide of the blood falls during each occlusion and rises again during each period of free flow of blood through the medulla until about forty per cent. of the total volume of the circulating blood has been drawn off for analysis. Beyond this point, there is a fall in the concentration of the carbon dioxide of the blood as each sample is drawn. Hemorrhage

| Character of Operative Procedure. | Amount of $CO_2$ Expressed in c.c. per c.c. of Blood Plasma. | Direction of Change in Total $CO_2$ of the Blood. |
|---|---|---|
| Etherization | .490 | |
| Two vertebrals and one carotid occluded. Blood pressure from other carotid. Ether intermitted | .525 | |
| One carotid released | .485 | |
| " " occluded | .460 | Fall. |
| " " released | .480 | Rise. |
| " " occluded | .435 | Fall. |
| " " released | .460 | Rise. |
| " " occluded | .445 | Fall. |
| " " released | .447 | 44 per cent. of total volume of blood drawn up to this time. |
| " " occluded | .380 | Rapid fall. |
| " " released | .350 | Slow fall. |
| " " occluded | .311 | Rapid fall. |
| " " released | .247 | Rapid fall. |
| " " occluded | .247 | Animal died. |

alone, after forty per cent. of the blood has been drawn will give a similar fall in the carbon dioxide of each successive sample of blood without occlusion of the remaining carotid artery.

## 31 (1406)

## The influence of milk upon tetany in Salamander larvæ.

By EDUARD UHLENHUTH

[*From the Rockefeller Institute for Medical Research, New York.*]

As reported in the meeting of the Society held on November 15, 1918, calcium lactate as well as magnesium lactate suppresses the tetanic convulsions of thymus-fed tetanic larvæ of salamanders. Since it has been claimed that milk also has this effect, it was interesting to test its action on tetanic larvæ.

Curves are demonstrated which show the percentage of tetanic individuals among two series of thymus-fed larvæ of the salamander, *Amblystoma opacum*. The animals of one series were kept in a weak milk solution, those of the other series which served as controls were kept in tap water. It is evident from the curves that milk was extremely effective in suppressing the tetanic con-

vulsions, since of six animals so treated only three suffered from convulsions (and these suffering only one attack), while the other three larvæ never showed convulsions at all.

But notwithstanding the favorable influence of milk upon the convulsions, milk like Mg and Ca salts did not prevent the development of permanent paralysis and permanent spasmodic contractions of the muscles.

Therefore, it must be pointed out again that the development of the paralysis of the muscles, in the presence of the salts and in the absence of convulsions, proves that tetany is due to a specific toxic substance which is not antagonized by calcium, magnesium, or milk. Furthermore, it appears that tetany (or at least some of its symptoms) is due to the toxic action of this substance upon the central nervous system, as indicated by the paralysis of almost the entire muscular system. How far these nervous lesions are responsible for the tetanic convulsions and how far the convulsions are due to the deficiency of calcium, remains to be determined.

32 (1407)

## The effect of heat, age and reaction on the antiscorbutic potency of vegetables.

By ALFRED F. HESS and LESTER J. UNGER.

[*From the Bureau of Laboratories, Department of Health, New York City.*]

The present communication is a continuation of experiments on antiscorbutics previously reported.[1] It was found that it required 35 gms. of the carrots used to feed our laboratory animals, to afford protection against scurvy to a guinea-pig. After the carrots had been cooked for three quarters of an hour, their addition to the dietary proved insufficient to protect. This was true even if the water in which they were boiled had been acidulated by the addition of 10 per cent. of vinegar. The only difference noted in the latter test was a less marked loss of weight.

A parallel test was carried out with carrots which had been picked only a few days previous to the experiment. It was found that, even subsequent to cooking, 35 gms. of these fresh carrots,

[1] PROCEED. SOC. EXPER. BIOL. & MED., XV., pp. 82; 141; XVI., p. 1, 1918.

when added to the dietary of hay, oats and water, were fully capable of protecting the animals. It is evident, therefore, that, in a consideration of vegetables as a foodstuff, we must take into account the factor of freshness. In dietetics this difference is intensified by the fact that older vegetables are tougher and therefore require and receive more prolonged cooking, thus further lessening their antiscorbutic value. The water in which the vegetables were cooked possessed little or none of the accessory factor, although 40 c.c. per capita were fed to the guinea-pigs; the animals did not, however, lose weight as rapidly as those receiving tap water.

In a previous communication it was shown that 5 c.c. of canned tomatoes is sufficient to protect a guinea-pig from scurvy. If such tomatoes are boiled for five minutes, their potency is slightly diminished, so that they should not be subjected to cooking when employed as an antiscorbutic for infants. Their efficacy was not diminished by rendering them slightly alkaline to phenolphthalein. Orange juice, which had been made $n/20$ alkaline to NaOH, was found to be just as potent as in the acid state. The tomato as well as the orange juice was given by a pipette one half to three quarters of an hour following alkalinization. Neither of these antiscorbutics, however, will retain their power long after they have been rendered alkaline. In judging of the effect of alkalinization or of heat, it is highly important to consider the length of time to which the antiscorbutic has been subjected to this influence.

33 (1408)

## Studies of saliva in its relation to the teeth.

### I. On the Normal Composition of Saliva.

### 1. *Does normal saliva contain uric acid (urate)?*

By **G. A. Lowenstein** and **William J. Gies.**

[*From the Biochemical Laboratory of Columbia University, College of Physicians and Surgeons, New York.*]

By the use of a slight modification of the Folin-Benedict method for the determination of uric acid in blood, we definitely established the presence of uric acid (urate) in saliva. The average

quantity of uric acid in saliva from men amounted to 2.10 mg. per 100 c.c. of the secretion; in saliva from women it amounted to 1.11 mg. per 100 c.c. We also succeeded in separating uric acid, in crystalline form, from saliva.

For normal individuals, the proportion of uric acid in saliva was independent of the diet, speaking generally, but was influenced by the rate of secretion as well as by the nature of the stimulant employed to accelerate the flow of the saliva.

Saliva appears to register promptly the variations in the endogenous metabolism of uric acid. We noted an almost immediate rise in the proportion of uric acid in saliva after increased muscular exertion, and after the ingestion of purine-free food following a brief fast. We also observed a definite relationship between the quantity of uric acid excreted in saliva, and the quantity eliminated simultaneously in the urine, in normal people on an ordinary diet.

The details of this study, and those related to it, will be published in the *Journal of Dental Research.*

34 (1409)

## The effect of large doses of X-rays on the resistance of monkeys to experimental poliomyelitis.

By **H. L. AMOSS, H. D. TAYLOR** and **W. D. WITHERBEE.**

[*From the Laboratories of the Rockefeller Institute for Medical Research, New York.*]

After a few passages through monkeys poliomyelitic virus becomes adapted and virulent for these animals. Hence slight variations in the susceptibility of these animals are not usually observed in experimental work. The original strain adapted to monkeys by Flexner and Lewis has been passed through many monkeys and stored in 50 per cent. glycerol in the ice-box, and its power to infect monkeys is very much diminished. This strain offers the opportunity of detecting variations in the susceptibility of the experimental animal to this infection. In two experiments an intracerebral injection of 1 c.c. of a Berkefeld filtrate of a 5 per cent. suspension of poliomyelitic cord containing the attenuated

virus produced typical experimental poliomyelitis in the monkey which has been exposed to large doses of X-rays (6 Holzknecht units daily for six or seven days), whereas in the non-rayed control no symptoms were observed. The doses of X-rays were sufficient to reduce greatly the number of circulating lymphocytes in the blood of the monkeys. In another experiment 0.75 c.c. of the filtrate produced typical experimental poliomyelitis in the X-rayed monkey, whereas 1.0 c.c. produced no symptoms in the control.

In an attempt to diminish an active immunity a monkey which had passed through an attack of experimental poliomyelitis and recovered with residual paralysis was exposed to large doses of X-ray so that the circulating lymphocytes were decreased from 27,000 per cu. mm. to about 2,500. Two separate intracerebral injections of active virus failed to produce any further symptoms or paralyses in this monkey.

35 (1410)

## Intrapulmonary irrigation.

By **M. C. WINTERNITZ** and **G. H. SMITH** (by invitation).

[*Department of Pathology, Yale School of Medicine, New Haven, Conn.*]

In view of the limitations of the intravascular mode of therapy of respiratory conditions, as exemplified in pneumonia, a series of experiments has been conducted looking into the possibility of an intratracheal or intrapulmonary therapy for such conditions.

The data thus far secured demonstrates the fact, fundamental to any such therapeutic procedure, that the lung is much less susceptible to the introduction of fluid than has been generally assumed.

Normal dogs have been used throughout the work and all perfusions or irrigations have been made with normal salt solution. The fluid was introduced by the usual method of insufflation.

Repeated experiments have shown that the lungs can be entirely flooded with salt solution and the process of irrigation continued for at least two hours with the introduction of 30,000 c.c. of fluid without causing any evident harmful signs in bodily well-

being or any subsequent serious microscopic lesions in the lung tissue.

By means of the use of colored solutions, such as India ink, it has been shown that even the last portions of the fluid introduced actually pass throughout the lung tissue and do not simply flow back through the trachea without entering the lung.

Such irrigation procedure is relatively effective in removing material from the lung. The preliminary insufflation of such materials as India ink, protargol, or starch paste, followed by irrigation with salt solution has shown that a large percentage of the indicator was removed by the perfusion. In the same way an irrigation of the lungs with 3,000 c.c. of salt solution following an insufflation of a heavy broth culture of *B. prodigiosus* removed 90 per cent. of the number of organisms introduced.

As a corollary to the above experiments a study was made of absorption from the lung. A series of dogs was given by insufflation 20 c.c. of salt solution per kilogram of body weight, and at stated intervals the dogs were killed and the lungs were examined grossly and histologically. The appearance of the lungs immediately after the insufflation showed them to be filled with fluid. Within 48 hours much of the fluid has been absorbed and after four days the lungs are practically free of fluid. Histologically, the lesions were inconspicuous, giving evidence of but little inflammatory reaction.

The rapidity of absorption was further confirmed by comparative determinations in the same animal of the excretion of phenolsulphonephthalein when administered by intramuscular and intravenous injections, and by intratracheal insufflation. The percentage excretion within two hours after the several modes of administration were:

| | |
|---|---|
| Intravenous injection | 78.1 |
| Intramuscular injection | 72.5 |
| Intratracheal insufflation | 57.1 |

# SCIENTIFIC PROCEEDINGS.

ABSTRACTS OF COMMIUNCATIONS.

**Ninety-seventh meeting**

*College of the City of New York, February 19, 1919.*

*President Gies in the chair.*

36 (1411)

**The effect of conjugation.**

By GARY N. CALKINS.

[*From Columbia University, New York City.*]

A single individual (ex-conjugant) of *Uroleptus mobilis* was isolated November 20, 1917. Five lines of the series were maintained by the usual daily isolation culture method used with infusoria. The relative metabolic activity is indicated by the average division rate of all five lines of the series for ten-day unit periods extending throughout the life cycle. Once a week, the excess individuals, after the isolations are made, are placed in larger culture dishes for a conjugation test. Here they multiply by division, until, in a week or ten days, thousands of individuals are present. With increasing scarcity of food they will conjugate, provided the protoplasm is sexually mature. From time to time, ex-conjugants, obtained from such conjugation tests, are isolated to form the beginnings of filial series. These are similarly maintained in daily isolation cultures, five lines to each series, and all fed at the same times and on the same standardized culture medium as the individuals of the parent series. This method furnishes the possibility of comparing the vitality of a filial series with that of the parent series. The protoplasm of 16 such series has been studied, each series represented by five lines; seven series have

died and nine are still under observation in different stages of vitality.

The process of parthenogenesis ("endomixis" of Woodruff and Erdmann) occurs in *Uroleptus* while encysted. These cysts require drying before the individuals will emerge. Parthenogenesis, therefore, is too clearly advertised to be overlooked, and the effects of such asexual reorganization cannot confuse the results obtained by the isolation cultures, for, while parthenogenesis occurs in the conjugation tests, it never has occurred in the isolation cultures.

In these experiments we deal with one protoplasm originally contained in the single-celled ex-conjugant of the A series. Some of this protoplasm has been maintained in isolation cultures where conjugation and pathenogenesis have been prevented; some is represented by protoplasm that has passed through processes of conjugation, both closely-related individuals in all cases being of the same age and having had the same identical treatment daily and with the same standardized food as the protoplasm of the parent isolation cultures; and some is represented by individuals that have passed through the processes of encystment and parthenogenesis.

The problems presented are: (1) Does the protoplasm of an ex-conjugant and its progeny by division, undergo progressive weakening of metabolic vigor ending in natural death? (2) Does conjugation between two individuals of the same age and each composed of similar, weakened protoplasm, result in the restoration of metabolic vigor to an optimum? The results of the experiments prove clearly, that both questions, so far as *Uroleptus* is concerned, are answered in the affirmative.

First, as regards physiological weakening and natural death. The original A series died in the 313th generation. The B series (obtained from a cyst) died in the 258th generation. The C series (from A 78) died in the 349th. The D series (from A137) died in the 271st. The F series (from C 86) died in the 317th. The G series (from B 115) died in the 291st, and the H series (from A 237) died in the 268th generation. All of the other series are still living, although two of them (I and J) will be dead within a month.

Using a longer unit period of 60 days to avoid the minor fluctuations of the 10-day periods, we find that all ex-conjugant series show a remarkable uniformity in corresponding periods. The standardized diet was not used until the 50th day of the parent A series so the rate for the first 60 days of the A series is not computed. The mean division rate, computed biometrically, for the first 60 days of the C series was 8.6333± .2185, or a rate of 17.26 divisions in 10 days; for the first 60 days of the D series, it was 8.58333± .1349 or 17.16 in 10 days; for the F series it was 8.600± .2320 or 17.2 in 10 days; for the H series it was 8.666± .1892 or 17.33; for the I series (from F143) it was 8.5833± .2468, or 17.16; for the J. series (from A 311) it was 8.966± .2252 or 17.93 and for the first 60 days of the L series (from I 199) it was 8.7000 ± .2986 or 17.4 divisions in 10 days. All ex-conjugant series, therefore, start with an initial optimum average division rate of 17.± divisions in 10 days. In the second 60-day period for each series, the rate falls to 15.± divisions in 10 days. In the third 60-day period the rate falls again to from 8 to 12 divisions in ten days while the variations become more marked. In the fourth 60-day period, the average division rate falls still lower, and all series, thus far, have died before the end of the fifth period. This decreasing vitality and death, cannot be due to food or other environmental factors, for, while one series is dying, a filial series whose protoplasm has been under identical conditions for the same length of time, is in full metabolic vigor. The cause of depression and death is endogenous, not exogenous.

Second, as regards the effect of conjugation on vitality. This question obviously is already answered by the results given above. In every case conjugation results in restoring the lagging activities to optimum metabolic vigor, indicated by the rate of 17 + divisions in 10 days, for the first 60-day period. It is still more obvious if we compare the metabolic activity of this protoplasm after it has conjugated, with a portion of the same protoplasm which has not conjugated. Thus the J series was derived from an ex-conjugant of the A series when the latter was in the 311th generation and nearly exhausted. The mean division rate of the offspring (J) was 17.9 divisions in 10 days while that of the parent (A) for the same calendar period, was only 0.25 divisions

in 10 days, a difference in vigor amounting to more than 17 divisions in ten days. In other words, if the two individuals, one of which formed the J series, had not conjugated, they would have had only enough metabolic vigor to divide at the rate of one division in 40 days, but, having conjugated, their metabolic vigor was such that they actually divided at the rate of 71.6 divisions in 40 days. This is an extreme case; if conjugation occurs before vitality runs so low, the difference between parent and offspring is less. Thus, the C, D and H series came directly from this same A series, C from individuals in the 78th generation, D from the 137th and H from the 237th generation, of the parent series. For the first 60 days of each filial series the mean division rates for ten days of offspring and parent were: C 17.26, A 15.73; D 17.16, A 14.13; and H 17.33, A 12.53, the differences being 1.53, 3.03 and 4.80.

Exactly similar results were obtained with the $F_2$, $F_3$ and $F_4$ generations of the original A series, some of which are dividing today with the optimum vigor of 17 + divisions in ten days.

Directing attention again to the fact that all series are treated in the same identical way as regards food and environmental conditions, and that conjugations were invariably between two individuals of the same age, the conclusion is incontestable that one fundamental effect of conjugation is the renewal of vitality, or rejuvenescence, of the protoplasm.

37 (1412)

## Endomixis and size variations in pure lines of Paramecium aurelia.

By RHODA ERDMANN.

[*From the Osborn Zoölogical Laboratory, Yale University, New Haven, Conn.*]

Jennings[1] considers the mean size of a pure line as strictly hereditary throughout the pure line; it belongs to one of the fundamental characteristics of this individual pure line. Eight years

[1] Jennings, H. S., *Proc. Amer. Philos. Soc.*, 47, 393–546.

later the same author[1] reverses his opinion and based on extensive studies, claims that a single stock, *i. e.*, a pure line derived by fission from a single progenitor gradually differentiates into such hereditarily diverse stocks; so that by selection marked results are produced. His former conception of the genotype was gained by experiments with the highly specialized infusorian species *Paramecium caudatum* and *aurelia*, his later ideas by his results with *Difflugia corona*, an amœbina of simpler cytological structure.

*Difflugia* has definite structural characters that can be counted and measured, which are unchanged by growth and environmental conditions, but still are "hereditable, yet variable." This coincidence of favorable conditions—besides theoretical considerations —gives a priori more support to views that do not maintain the absolute constancy of the genotype, though they contradict the current conception of the "so-called" pure line. Paramecium is changed in size by its daily divisions and by the influences of the environment, thus complicating genetic studies; Jennings's results have been challenged by Walton[2] and Castle,[3] as statistically considered far from conclusive. The mean length, a constant after his opinion, varies between 114.033, 123.606, 130.120 and 144.880 microns for mass cultures, derived from the same animal. A new complication arose after the discovery of endomixis,[4] a dynamic, periodic reorganization process, involving the disintegration and absorption of the old macronuclear and micronuclear material without the introduction of foreign nuclear material. It was evident that the discrepancies in Jennings's measurements might be explained, if there are periodically appearing fluctuations during the intermictic periods. Further, before the influence of conjugation in *Paramecium* can be studied, it is necessary to know first the isolated influences of division, clearly shown by Jennings and reinvestigated for the lines I worked with, to eliminate the changes produced by environmental conditions and the most difficult process, to find, if endomixis has influences on the variability of the quantities of the line. These are the preparatory

[1] Jennings, H. S., *Genetics*, 1, 407–534.

[2] Walton, L. B., *American Naturalist*, 49, 642–652.

[3] Castle, W. E., *American Naturalist*, 50, 179–183.

[4] Woodruff, L. L., and Erdmann, Rh., *Jour. Exp. Zoöl.*, 17, 425–516; Erdmann, Rh., and Woodruff, L. L., *Jour. Exp. Zool.*, 20, 59–96.

steps to be taken, before attempting to decide, if conjugation has an effect and which particular effect in *Paramecia*. All authors till now have studied the combined influences of unrecorded occurrences of endomixis and influences of recorded occurrence of conjugation. The intermictic period of 28 days duration alternates with the endomictic phase indefinitely. The climax, when the most important steps of the reorganization process take place, lasts one or two days, while the ascending and descending phases of the endomictic period overlap either with the first days or last days of the intermictic period. The number of daily divisions amounts to one or two, hence approximately 50 in one intermictic period, which we have divided for convenience into three subperiods. Changes effected by the constantly occurring divisions and by the approaching of the next endomixis go parallel and can not be considered separately.

By our culture methods we can create a constant environment, by previous observations we can recognize how a culture changes in mean length and mean breadth, standard deviation and correlation between length and breadth before, in and after division. We eliminate further by our methods all animals while dividing or in constriction and then we are able to recognize periodical regular combinations of the four quantities in question in each intermictic subperiod. In the second subperiod the animals reach their mean length, have a medium to high standard deviation and correlation; in the third subperiod they do not reach their mean length, but have a very high standard deviation and correlation. In glancing over the changes in the quantities shortly after or in division, we know that the four quantities in question change too in value, *i. e.*, the standard deviation is low immediately after division, and high shortly before division. Therefore in each intermictic period the values of the four quantities are in the whole measurements either balanced, augmented or diminished. We find that a culture shortly before the onset of the next endomixis is distinguished by a low division rate, a low mean length, a high mean breadth, a high standard deviation and high coefficient of correlation. In Line I the standard deviation passes through the following values during the intermictic period, Line I $5530_{10}$ with 2.424 +. 105, Line I $5530_{14}$ with

3.593 + .156, and Line I $5530_{24}$ with 4.259 + .185 units for the different mean lengths in each different subperiod. These changes appear in each investigated line, Line I, Line IE and Line O, in each intermictic period, thus affording opportunity to select the best period for deriving comparable values in our subsequent measurements. When, after 14 days the animal of known number of generations is transferred from the isolated slide culture into the medium and is allowed to multiply, the mass culture is best for our purposes: it is on the height of the second subperiod of the intermictic period, when the culture as a whole attains its highest mean length.

In the mean lengths of subsequent measurements for the same Line IE from 1914 till 1917, covering 2,200 generations in the life history of this line, the maxima cluster all around the same number 105 microns or 30 of our units of measurement.

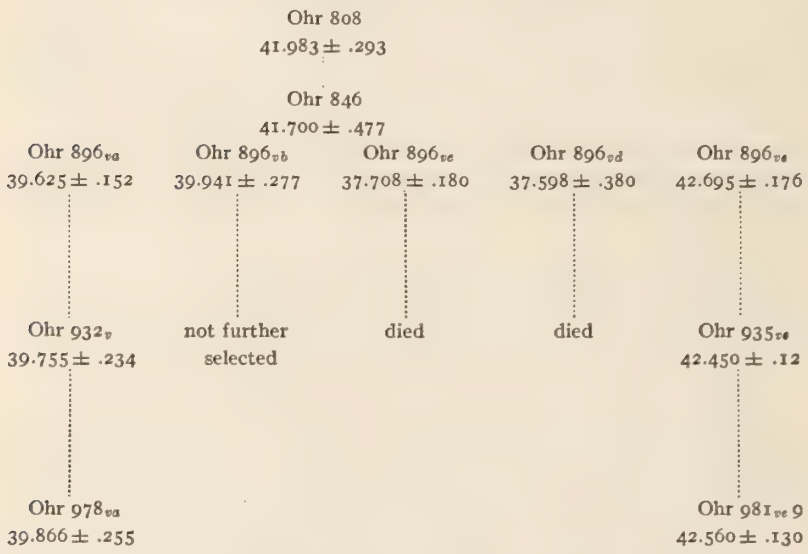
Line I, from which was derived at the 4,020 generation the above mentioned line IE, has a mean length of 112 microns or 32 units, another line O that was only recently in laboratory culture, and in which endomixis was observed from its occurrence in 179 till the 901 generation had a mean length of 41 units or 143.5 microns.

The periodical change in all dimensions proceeds while the culture passes the intermictic period, then endomixis occurs, effaces the high standard deviation and coefficient of correlation and a culture with low quantities appears in the first subperiod of the intermictic period, reaching later in the second subperiod the theoretical mean length and showing "constancy" of this dimension for years under the same environmental conditions. Endomixis therefore acts as a *stabilizer* and effaces the fluctuations around the mean, that Jennings had seen in his cultures.

But endomixis also acts as an *originator* of new lines with a mean length of their own. If the division products of the two divisions after endomixis are seeded out and the progeny of these eight lines measured, these lines show different mean lengths, though bred in the same conditions and closely related. Only five animals produced mass cultures, the measurements are given on the following table.

If these lines are carefully carried through the next endomictic

LINE OHR AFTER OCCURRENCE OF ENDOMIXIS IN THE 896TH GENERATION IN ITS BREAKING UP PROCESS; ONLY MEAN LENGTHS IN THREE SUBSEQUENT ENDOMICTIC PERIODS ARE GIVEN IN UNITS.
(One unit = 3.5 microns.




period and the selection for a certain mean length repeated, it is possible to isolate lines with different characters. The table shows the measurements for two cultures, carried through three subsequent endomictic periods, derived both at the 896th generation, these lines Ohr $896_{va}$ and Ohr $896_{ve}$ remain "constant." The same phenomenon was unintentionally attained when during our joint studies, Dr. Woodruff and I kept for a long sequence of generations Line IE isolated from Line I, the famous Line already under laboratory observation since 1907. The breaking up in "hereditarily diverse stocks" in this line could not be attributed to the supposed effects of the culture methods that varied only for about 800 in the total 2,200 observed generations, for both lines. I cultivated Line O, either in hay infusion or bouillon, either in room temperature or in constant temperature. The dimensions in the beginning show a more or less marked decrease, but later come nearly to the old standard. Such a considerable difference as Lines I and IE or Lines Ohr $978_{va}$ and Ohr $981_{ve}$ show and maintain, can not be affected by environmental changes.

The production of new lines is caused by endomixis, the reorganization process in paramecium, so closely related to the sexual phenomenon of conjugation and still so essentially different from this process by being effected in one cell and not affording the introduction of foreign chromatin into the cell. We have to conclude that even in so called "asexually" propagated lines heritable variations can be produced by directed selection. But why do these new lines not oftener appear in nature or in the laboratory? First different observers have given different measurements for strains of *Paramecium*, isolated from populations; Jollos reports the breaking up of a line, and Jennings the appearance of lines with new heritable characters after conjugation. Owing to the difficult culture conditions he could, at that time, not prove definitely that the breaking up in different new lines is effected by conjugation. Now it is possible to do so. But the rôle of the organization process in question is twofold: Endomixis gives rise to new combinations that can be selected, but also stabilizes the line at each occurrence. If variations in mean length appear between 37 and 45 units, those lines will thrive best either in mass cultures or isolated lines, which can adapt themselves the quickest to the given environment. I have observed that all descendants of the O lines with 37, 43 and 45 units die in the constant environment I choose for all these experiments. The 41 mean length and 11 mean breadth had the best chances to survive in the same environment. The uniformity, a word that should be better used than constancy in the appearances of the genotype, is a product of the influences of the chosen environment and of the adaptability of the different recombinations appearing after endomixis. These recombinations will become "heritable," if the environment remains nearly constant, else other recombinations appear after endomixis that are better adapted to the new conditions.

What specific effects conjugation, the other reorganization process, has in the life of *Paramecium* is still unknown.

[6] Jollos, V., *Biol. Centralbl.*, 33, 222–236.

38 (1413)

## Electrical stimulation and $CO_2$ production in nervous tissue.

By A. R. MOORE.

[*From the Physiological Laboratory of Rutgers College, New Brunswick, N. J.*]

With his barium carbonate precipitation method, Tashiro[1] found that the $CO_2$ output of a frog's nerve more than doubled when stimulated with induction shocks for 10 minutes. In order to avoid certain objections to Tashiro's method, viz., heating effect of the current,[2] and death changes due to the drying of the tissue, the indicator method previously described[3] has been employed. This method permits the tissue to be frequently or even continuously bathed with the Ringer's solution during the experiment. Stimulation was obtained by means of platinum electrodes passed through the cork closing the test-tube, and bent in the form of hooks so that they served as holders. The secondary coil of the Harvard inductorium stood at 12.

Small strips of the sartorius muscle of the frog served as controls. During stimulation with the tetanizing current such a strip was allowed to contract isotonically. In this condition the muscle showed no increase in the rate of $CO_2$ production, but upon relaxation, immediately following stimulation, the rate was approximately doubled. With sciatic nerves and the medulla, however, it was not found possible to produce any significant change in $CO_2$ production of the tissue with 1–2 minutes stimulation.

[1] Tashiro, S., "A Chemical Sign of Life," Chicago, 1917, p. 38.

[2] Lucas, K., "The Conduction of the Nervous Impulse," London, 1917, p. 26.

[3] Moore, A. R., PROC. SOC. EXP. BIOL. AND MED., vol. 16, pp. 35–39, 1918.

39 (1414)

## Mesenchymal activity as a factor in resistance against mouse sarcoma in chick.

By VERA DANCHAKOFF.

[*From Columbia University, George Crocker Special Research Fund, New York City, F. C. Wood, Director.*]

The digestive capacity of a mesenchymal cell in the embryo and of a connective-tissue cell in the adult organism has been recorded many times in the literature.

A mesenchymal embryonic cell, being a very mobile element, will easily detach itself from the common mesenchymal syncytium, and in the presence of a foreign body will ingest it. Under normal conditions, such foreign bodies in the embryo are for the greater part red blood-corpuscles. While the vascular channels in the embryo are undergoing extensive rearrangement, erythrocytes are frequently found free amongst mesenchymal cells and ingested by the latter. Whether or not an erythrocyte undergoing ingestion is still alive, we do not know. It is a highly differentiated cell, in an unfavorable medium while outside the vessels, and with no further power of proliferation.

The ingested blood-cell undergoes within the phagocyte (of mesenchymal origin) a series of chemical changes, some of them demonstrable under the microscope, which transform it into a structureless mass of protein and result in complete digestion. The embryonic mesenchymal cell, therefore, not only is able to synthesize proteins at the expense of amino-acids, but has itself a digestive power. The intra-cellular digestive capacity of a mesenchymal phagocyte may give us a basis for the understanding of other aspects of a similar activity. Thus, chondroclasts, osteoclasts, and clasmatocytes, which are but modified mesenchymal cells, exercise a digestive power, either intracellular or extracellular.

The mesenchymal embryonic cell is capable of digesting its own proteins in the form of dead cells and possibly some living cells which have lost their normal correlations with the tissues of the

organism, and it exercises this power from the earliest stages of embryonic development. We know little of its response to foreign proteins and to normal living cells. We do know, however, that it has no power over, and, therefore, no injurious effect upon, tumor cells. Tumor cells will grow amongst, and together with, mesenchymal cells.

The digestive power of adult mesenchyme (fibroblasts, clasmatocytes, splenic cellular reticulum) is much greater. We see fibroblasts and clasmatocytes ingest red blood-corpuscles after hemorrhage, and particles of a disintegrating nerve after section of a nerve trunk. Under normal conditions macrophages in the spleen are actively engaged in the phagocytosis of red blood-corpuscles. Artificially separated cells of the splenic mesenchyme may contain dozens of red and white blood-cells in their cytoplasm; or, again, fibroblasts in a culture may be seen loaded with particles of artificial medium. The ingested substances undergo an intracellular digestion.

Moreover, the adult mesenchyme of the splenic cellular reticulum, when compared with that of the embryo, is found to have acquired a new property. Embryonic splenic mesenchyme in the chick does not show any inhibiting power even as late as the hatching period, much less any destroying power over any kind of tumor cells. Tumor and splenic mesenchyme of a hatching chick thoroughly mixed will grow on the allantois well, as though transplanted independently. Splenic mesenchyme of the adult fowl, on the contrary, possesses the power of checking the Ehrlich mouse sarcoma (in its present phase of growth in the Crocker Laboratory), and in retarding the growth of the very malignant mouse sarcoma 180. The photographed preparations (demonstrated by lantern slides), show a curious relationship developing between the mesenchymal part of the adult tissue and the tumor when these are thoroughly mixed together and grafted on the allantois of a 7- or 8-day chick embryo. The tumor cells are not injured mechanically by this procedure, nor do they show any signs of an immediate injurious action by the enzymes which are known to be present in the spleen, for intensive growth of tumor mixed with spleen is observed during the first two, and sometimes three days of further incubation of the egg.

The tumor begins to grow in such grafts in the form of small foci surrounded by adult mesenchyme, the tumor cells assuming the form of polygonal or fusiform bodies in a syncytial and even plasmodial arrangement. Soon, however, in the region in which both tissues come into contact, the tumor cells, whether in mitosis or in the resting stage, become separated one by one from the syncytium. Mesenchymal cells closely encircle them and form around them a wreath of nuclei with a common cytoplasm, frequently giving the impression of large giant cells with a tumor cell within their cytoplasm. The tumor cell, at first closely surrounded, is soon found to be situated in a vacuole, the cell itself diminishing in size, gradually losing its structure, and finally completely disappearing. A graft of Ehrlich sarcoma, though showing at first an extensive growth, and in control animals reaching in 7 to 9 days a size of 1 to 1.5 cm. in diameter, is generally brought by this process to disappear five days after grafting. Not a single tumor cell could be discovered under the microscope in full series of grafts, though the identification of the large cells of the Ehrlich sarcoma amongst the chick mesenchyme is rather easy. An absolute biological proof of the destruction of the tumor cells could be obtained by the inoculation of such grafts into mice. A similar activity of the adult splenic mesenchyme, though affecting sarcoma 180, was not sufficient to check completely this very fast-growing tumor, and the tumor still grew in spite of a partial destruction easily demonstrable under the microscope.

The process of separation of the tumor cells with their subsequent death and final disappearance of digestion, takes place on the whole circumference of the tumor foci, if tumor and spleen are thoroughly mixed together. If grafted separately but adjacent to each other, the process develops only in that region in which both grafts come into contact, and not on other parts of the circumference. If the grafts be separated by a considerable space, the reaction will develop if, and at the time when, both tissues come together. This reaction, therefore, depends not upon a resistance of the host, which was supposed to be conferred by the introduction into its organism of a bit of spleen with its small lymphocytes, but upon the functional properties of the splenic mesenchyme introduced. This process is certainly not a mechan-

ical result of intergrowth of tumors and any kind of mesenchymal tissue, for intergrowth takes place between the cells of the Ehrlich sarcoma and mesenchyme of a hatching chick with no injurious effect upon the tumor. The process depends, therefore, upon a property which the mesenchyme acquires after birth. There is a biological functional difference between the embryonic and adult splenic mesenchyme in the chick, apparent in its different response to the living tumor cell of the mouse sarcoma employed in these experiments. The functional capacity of the adult splenic mesenchyme—new in its power to injure a living tumor cell—might in my opinion be induced by factors closely connected with the great changes which take place after birth in all organs of digestion and assimilation.

40 (1415)

## Further proof of the antagonism existing between the thymus and parathyroid.[1]

By EDUARD UHLENHUTH.

[*From the Rockefeller Institute for Medical Research, New York City.*]

Larvæ of the salamander *Amblystoma opacum* when fed on thymus soon after hatching develop tetanic convulsions at an age of from 35 to 40 days. Since at this time the larvæ develop, in their own thymus glands, the structures characteristic for the secretory stage of the glands, it was concluded that the amphibian thymus like that of the mammalian thymus excretes a toxic substance producing tetanic convulsions, and that tetany results if the animal's own secretion is added to that introduced by the thymus diet.

This is confirmed by further experiments (Table I., first four horizontal rows), which show that the interval between the beginning of the thymus feeding and the outbreak of tetanic convulsions becomes shorter, the later the thymus feeding is started, while the age at which tetany develops, remains constant.

If thymus feeding is started after the development of the functional stage of the animal's own thymus glands has taken

[1] *Jour. Gen. Physiol.*, 1918, i., p. 23 and 33.

place, tetany develops as soon as a certain amount of the tetany toxin has been accumulated in the organism (Table I, Series XXV, 1918). The time required for accumulation of that amount is far shorter than the intervals in the first two series. It is, however, longer than in the fourth series, because the animals of series XXV. had been fed twice as long on normal diet and besides had almost twice as much time to grow before the thymus diet was started, and larger animals need more of the tetany toxin than small ones to develop tetany.

Hence, it is evident that the amphibian thymus gland manufactures a secretion similar to that of the mammalian thymus, and that the amphibian organism even in the absence of parathyroid glands can antagonize a certain amount of the toxin, be it excreted from its own thymus glands or introduced by thymus feeding before the animal's own thymus glands have developed; but excretion of their own thymus glands and thymus feeding at the same time lead finally to the accumulation of an excess amount of the tetany toxin, of which the parathyroidless organism of the salamander larva cannot dispose, and consequently tetany results.

But when the thymus-fed larvæ metamorphose, the tetanic convulsions stop and never recur after metamorphosis has taken place, even if the animals are continued on an exclusive thymus diet. Since during metamorphosis the animals develop their parathyroids, it was concluded that the parathyroid glands now serve to antagonize the tetany toxin and that the parathyroidal mechanism is more efficient than the one existing in the larvæ since the parathryoids are able to antagonize not only the tetany toxin excreted by the animal's own thymus glands but also the toxin introduced by the thymus died.

But it might be that after the tetany toxin has acted for a certain length of time upon the central nervous system and all the motor nerve cells have been destroyed, no further muscular contraction would be possible, and that possibly the time of complete destruction of the motor nerve cells coincides with the period of metamorphosis.

If this were true, one would expect that when a normal salamander is fed on thymus shortly after metamorphosis, tetanic convulsions would be produced after about four weeks of thymus

feeding, this being the time required to produce tetany in the larvæ, and that the tetany would cease, as in the larvæ, about 8 to 10 weeks after its commencement.

RELATION BETWEEN TETANY IN THYMUS-FED LARVÆ OF *Amblystoma opacum* AND THE DEVELOPMENT OF THE LARVAL THYMUS-GLANDS.

| Series. | Age at the Beginning of Thymus Feeding. | Age at the Beginning of Tetany. | Time Required to Produce Tetany. |
|---|---|---|---|
| XVI., 1918..... | 9 days. | 35 days. | 26 days. |
| B., 1916..... | 16 " | 39 " | 23 " |
| B., 1917..... | 22 " | 35 " | 13 " |
| T., 1917..... | 26 " | 35.5 " | 9.5 " |
| XXV., 1918..... | 45 " | 57 " | 12 " |

In order to decide this point, larvæ of the salamander *A. opacum* were fed on a normal diet which was continued for some time after metamorphosis had occurred. Finally three of these animals were put on an exclusive thymus diet. They have been fed now for seven months on a diet consisting only of thymus, but none of the animals has shown any signs of tetany, either of convulsions or paralysis of the muscles, while in the larvæ, tetany develops after several weeks of thymus feeding. Two of these animals together with a worm-fed control specimen are shown.

This finding appears to offer further proof that the ending of the tetany in thymus-fed larvæ at the time of metamorphosis is actually due to the development of the parathyroids at that time, and that the parathyroids are capable of antagonizing the tetany toxin contained in the thymus. Consequently tetany resulting from thymus feeding in the salamander larvæ is a true parathyreoiprival tetany.

41 (1416)

**The behavior of certain digitalis principles in the body.**

By ROBERT A. HATCHER and CARY EGGLESTON.

[*From the Laboratory of Pharmacology of Cornell University Medical College, New York.*]

The authors presented an outline of their method of estimating the absorption, destruction and elimination of several of the digitalis principles in the rat, and that of ouabain in the cat, with the results of some of their experiments.

42 (1417)

## The relative importance of the intestine and kidneys as excretory channels.

By VICTOR C. MYERS and MORRIS S. FINE.

[*From the Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital, New York City.*]

Despite the fact that during the past twenty years a number of investigators have attempted to dispel the popular notion that the intestine, in comparison with the kidney, is relatively unimportant as an excretory channel, this rôle of the intestine would not appear to be properly appreciated.

In connection with a study of metabolism in pellagra,[1] which we made several years ago under the auspices of the Thompson Pellagra Commission, fairly complete analyses of both the urine and feces were carried out. Thirteen subjects were studied. A lacto-vegetarian diet was employed, the experimental period extending from seven to ten days. Data on the water, nitrogen, sulfur, chlorine, phosphorus, calcium, magnesium and potassium outputs were obtained on both the urine and the stools, furnishing an interesting comparison of the kidneys and intestine as excretory channels. It is not believed that the findings differed especially from the normal, except in that group of cases which suffered from intestinal diarrhea. The average findings in five cases with well-formed stools, 74 to 79 per cent. moisture, and those with diarrheal stools, 79 to 89 per cent. moisture, have been grouped separately in the table below.

| Number of Cases. | Moisture Content of Feces, Per Cent. | Fecal Output in Per Cent. of Total Output of Both Urine and Feces. | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | $H_2O$. | N. | S. | Cl. | P. | Ca. | Mg. | K. |
| 5 | 76 | 6 | 10 | 10 | 3 | 36 | 90 | 72 | 18 |
| 9 | 84 | 16 | 15 | 19 | 9 | 33 | 89 | 68 | 27 |

An inspection of the table shows that in the first group of cases the total nitrogen and total sulfur parallel each other very closely, as probably might be expected from their common origin

[1] Myers and Fine, *Am. Jour. Med. Sc.*, 1913, cxlv, 705.

(protein). With diarrhea sulfur does not appear to be quite as well absorbed as the nitrogen. Although normally very little chloride is eliminated by the intestine, the amount found in the stools may be considerably increased in diarrhea. About one third of the total phosphorus output of the intestine and kidney is found in the stools. The percentage output in the feces of both calcium and magnesium is high, due, as we believe, to the lacto-vegetarian diet, which resulted in a poor absorption of compounds of these elements. As might be anticipated from our knowledge of potassium salts, a very appreciable amount of this element is eliminated in the feces, and diarrhea considerably accentuates this elimination. In general it may be said regarding the intestinal diarrhea, that, although it very definitely reduces the absorption of nitrogen, sulfur, chlorine and potassium, it appears to be almost without influence on the phosphorus, calcium and magnesium.

43 (1418)

## The pigment changes in frog larvæ deprived of the epithelial hypophysis.

By **P. E. SMITH** (by invitation).

[*From the Anatomical Laboratory, University of California.*]

It has been known for a considerable time that in the fishes, amphibia and reptilia remarkable changes in color pattern or external appearance occur. It is also generally known that these effects are due to the reciprocal interplay of alterations in at least two great systems of pigment-bearing cells, the bearers of dark pigment (melanophores) and the bearers of various other pigments, many of them metallic lustered (xantholeucophores). To the latter class of pigments the trout owes its silvery appearance.

Somewhat over two years ago the author, followed shortly by B. M. Allen, showed that peculiar silvery frog larvæ were invariably produced when the epithelial portion of the hypophysis was removed in early embryonic stages. For the sake of brevity these individuals were designated "albinos" and are always in conspicuous contrast to the darker, normal specimens. As might

be expected from our introductory remarks, both of the great groups of pigment-bearing cells contribute to produce this strange effect; but no mention of the iridescent cells (the xantholeucophores) was made by the writer at the time of his early communications on this subject[1] and it is noteworthy that in both of the subsequent papers by B. M. Allen[2] and in the recent communication by W. J. Atwell[3] these cells have received no attention whatever.[4]

It can be demonstrated easily that the silvery, or albinous, condition, as is the case in so many instances of the color change in animals, is participated in by both groups of cells, melanophores and xantholeucophores. Furthermore, it is indeed a fact that under normal conditions no change in the condition of one of these sets of cells takes place without a reciprocal alteration in the other. This conception was forced home to the writer not merely by a reëxamination of the anatomical causes for the albinos, but by extensive physiological and pharmacological experiments on normal and albinous specimens. It is also a fact that pigment changes, other than those associated with true pigment cells, have escaped the notice of most observers, although the writer demonstrated and commented upon the conspicuous reduction of the superficial "free" pigment possessed by the epithelium at the time of his communication to the American Association of Anatomists in December, 1917. Since Atwell has raised the whole

[1] "Experimental Ablation of the Hypophysis in the Frog Embryo," *Science*, N. S., vol. 44, no. 1130, August 25, 1916 and "The Effect of Hypophysectomy in the Early Embryo upon the Growth and Development of the Frog," *Anatomical Record*, vol. 11, October, 1916.

[2] "Extirpation Experiments in *Rana pipiens* Larvæ," *Science*, vol. 44, November 24, 1916, and "Effects of the Extirpation of the Anterior Lobe of the Hypophysis of *Rana pipiens*," *Biological Bulletin*, vol. 32, no. 3, March, 1917.

[3] "On the Nature of the Pigmentation Changes Following Hypophysectomy in the Frog Larvæ," *Science*, N. S., vol. 49, No. 1254, January 10, 1919.

[4] That a satisfactory analysis of the condition of these cells has not hitherto been made may perhaps be attributable to two causes, first, the fact that the tail is atypical in this respect, and it would appear that Atwell on account of the advantage of employing Clark's beautiful mechanism for observing the living tail has paid too much attention to this locality; secondly, the fact that the xantholeucophores lose their pigment content and are hence impossible to detect in sections after many fixing fluids. Formalin, Zenker's fluid and Bouin's fluid, for instance often produce this effect after a short interval, although Helly's fluid and some other chrome mixtures fortunately preserve them.

question in his recent paper, it seems desirable, in advance of a more extensive presentation, to describe briefly the various anatomical findings which it can now be stated underlie the albinous condition.

The study of great numbers of such larvæ produced by experiment during the last three years, conducted, it is to be emphasized, both on living and on appropriately fixed specimens, has given uniformly concurrent testimony that these hypophysis-free albinos are produced by three chief alterations of the pigment mechanism. These may be enumerated as, (1) Reduction in the system of epidermal melanophores, consisting of greatly lessened numbers of these cells and in the contracted and pigment-poor condition of those cells which are present; (2) a marked reduction in the number of so-called free pigment granules of melanin in the epithelium; (3) an invariable expansion of the xantholeucophores situated in both deep and superficial strata of the dorsum of both head and body.

The condition of the melanophores is especially interesting, due to Atwell's contention that previous investigators have overlooked what he feels to be the major contribution towards the albinism made by a contraction of the subepidermal melanophores. Atwell bases his contention on three lines of evidence: first, an increase in the pigmentation, producing almost a normal depth of color, when albinous larvæ are treated with a solution of dried pars intermedia substance; secondly, the preparation of many whole mounts of the skin of albinous larvæ in which he claims to have discovered an invariable contraction of the deep melanophores; thirdly, observations on the living tail fin of albinous larvæ treated with an extract of pars intermedia where an expansion of these cells was observed. It is only fair to state that full admission is made of the reduction of the epidermal melanophores, though this is rated as of secondary importance. Albinous larvæ have never in the hands of the writer been appreciably increased in the depth of pigmentation even with the use of the one procedure most potent in expanding the subepidermal melanophores—the sunlight. Moreover, it is extremely difficult to understand Atwell's contention that the contraction of these cells is the main cause of albinism when we are confronted with the anatomical

relationships involved in all the main body area, aside from the ventral region, for a layer of xantholeucophores intervenes between the epidermis and the subepidermal melanophores, a layer which in the albino so completely screens off the subepidermal melanophores as to make their observation in life extremely difficult. It will readily be understood that this effect is greatly emphasized by the great expansion of the xantholeucophores which the author has invariably found in albinism.

As regards the second point, it is necessary to remark that every precaution must be taken (temperature, background, speed of fixation, etc.) in the preparation of material for study in order to justify any certainty that the condition of the pigment cells has not been changed by a complicating extraneous factor. Comment has already been made on the impropriety of applying observations made on the tail fin to the very differently constituted skin of the dorsum of the body. Moreover, it is not to be wondered at that an expansion of the subepidermal melanophores could be observed after the application of pars intermedia extract to animals in a Clark chamber where the strong illumination is probably alone sufficient to produce this result.

In the experience of the writer no constant deviation exists from the various states of relaxation or expansion which may occur normally in the subepidermal or deeper melanophores of albinos as contrasted with normal larvæ.

There is no need to comment on the writer's contention that the epidermally situated melanophores of albinos are greatly at fault; both subsequent observers have confirmed him in the great reduction of the number of these cells; and Atwell has observed the reduction in the pigment content of the remainder, which appeared to have been denied by Allen. Though we have felt free to contest the view of Allen that a contraction pertains in the deep melanophores of the body, corroboration must be given to his views as pertains to the physiological state of the epidermal melanophores. The epidermal melanophores are scanty in number, reduced in pigment content, and most of them exhibit varying degrees of contraction.

As regards the great reduction in the so-called free pigment in the superficial layer of the epidermis, the writer wishes to reiterate

his statements made in 1916. While it is impossible to attribute more than a minor rôle to this, nevertheless, it is just as striking and constant as the other pigment effects. This alteration in the epithelial "free" pigment would appear to have escaped detection along with the decided changes in the xantholeucophore cells. Attention has already been called to the solvent action of many fixatives on these cells. Their identification is not interfered with by the use of Helly's fluid and is rendered easiest of all when whole mounts preserved in this way are explored with the polariscope with which the doubly refractive powers of the guanin substance is brought out. It is proper here to call attention to the fact that the maximal expansion which these cells enjoy in albinous larvæ can also be overlooked because of a subsequent contraction in them which occurs with the use of anesthetics and in many conditions of impaired vitality occurring through disease or intentionally experimentally produced as with too strong doses of adrenalin. In another place comment will be made on the change in the physiological and pharmacological reactions of both types of pigment cells; but it may be stated here that the widely expanded xantholeucophores of albinos are singularly unamenable to most experimental influences and in this they are in striking contrast to the iridescent cells of normal animals. That the expanded xantholeucophores contribute decidedly to the albinous appearance is shown by the behavior of those albinous larvæ fed on posterior lobe substance. These animals exhibit a partial recovery of the melanin deficiency which may indeed approach the normal. In spite of this, they are always conspicuously lighter than their normal controls, a fact readily explainable by failure of this treatment to influence the persistent expansion of the iridescent cells.

44 (1419)

**On the reaction of the pigment cells in normal and albinous frog larvæ.**

By **P. E. SMITH** (by invitation).

[*From the Anatomical Laboratory, University of California.*]

In the experimentally produced albinous frog larvæ which follow a successful early extirpation of the epithelial portion of

the hypophysis, a pigmentary system is produced which is not only strikingly different from the normal in its anatomical and physiological condition but also exhibits striking departures from the normal in its physiological responses.

Normal larvæ kept in diffuse light and on an indifferent background usually show a fully expanded, or only slightly contracted, condition of the epidermal melanophores[1] and a completely contracted, or but slightly expanded, condition of the corial xantholeucophores. This condition can almost always be somewhat exaggerated by submitting the larvæ to the simultaneous action of low temperature and darkness, when the epidermal pigment cells are fully expanded and the corial xantholeucophores minute, silvery dots. When such larvæ, or those from an indifferent environment, are submitted to reverse conditions, *i. e.*, the simultaneous action of warmth (33°–35° C.) and sunlight, these two classes of pigment cells react in the reverse way and in from one half to one hour exhibit a picture of contracted melanophores and widely expanded silver cells. It is thus seen that the reactions of the two groups of superficial pigment cells—the epidermal melanophores and the corial xantholeucophores—go hand in hand and are in a reverse direction.

Attention has already been called to the constant great expansion of the corial xantholeucophores in the albinos, an expansion which exceeds considerably that which can ever be obtained by the action of sunlight and heat on normal larvæ. Moreever, it is difficult to influence by physiological means this great expansion of the xantholeucophores in the albino. On the other hand, the epidermal melanophores of the albinos, which exhibit various stages of contraction, are widely expanded by the action

[1] This condition is the same as that characterizing the deep melanophores which harmonize in their behavior under exaggerated conditions of light and temperature with their more intimate associates, the xantholeucophores. The reactions of the deep melanophores have been the object of considerable study and in the experience of the writer are identical in albinous and normal individuals. Attention must again be called to the necessity in all experiments of this type of not merely submitting the animals and their controls to identical conditions, but also to start with a known physiological condition which has been produced by the action of a practically constant environment over a considerable period of time preceding the experiment, the effect of which upon the larvæ is known through careful examination of the living and unanæsthetized specimens immediately preceding the tests.

of heat and light, an effect just the opposite of that which the same factors produce in the normal skin.

Finally it may be mentioned that when all stimuli are removed the resulting condition of the pigment cells does not differ greatly in normal and albinous larvæ. A study of animals which have recently died in the aquaria, or have been purposely killed, shows that the pigment cells in both normal and albinous individuals come to approximately the same condition, which is one of partial expansion of the xantholeucophores and an expansion of the epidermal melanophores greater than can be produced by the action of light or heat or other experimental means in living animals. The reaction of the xantholeucophores in albinous and normal larvæ is also identical when subjected to all the anesthetics tried by the writer (paraldehyd, chloretone, and ethylurethan). In both they are greatly contracted.

45 (1420)

## Upon the experimental exchange of skin transplants between normal and albinous larvæ.

By **P. E. SMITH** (by invitation).

[*From the Anatomical Laboratory, University of California.*]

If sufficient speed is exercised, an exchange of the middorsal area of skin can be successfully accomplished between two frog larvæ, a normal one and its albinous mate. Since there are striking and constant differences between two such larvæ as concerns both classes of superficial pigment cells, a highly interesting opportunity to test the influence on such cells of a new host was presented. Four such successful skin exchanges were accomplished and in all instances definite and constant changes in the condition of the xantholeucophores were produced as a result of the exchange. The changes, which are well under way in an hour after such an experiment and which have yielded harmonious results, would appear to be of great value in the interpretation of the change in the physiological state of these cells which albinism produces. It will be recalled that the corial xantholeucophores

in hypophysis-free albinos present a greatly expanded condition and that this expanded condition is not amenable to most experimental influences (temperature and light) although anesthetics affect it. An exactly opposite physiological state of these cells, *i. e.*, a contracted condition, usually occurs in the normal larvæ. *Successful skin exchanges altered the state of the xantholeucophores to correspond to that characterizing the new host.* The change is usually observable within fifteen minutes and is invariably complete within four hours. Inasmuch as the change is much more pronounced than that exhibited by animals of weakened vitality or immediately after death, it can hardly be referable to merely a transient condition of weakened vitality. More especially is this the case, since the changes taking place terminate only when the state of the transplanted xantholeucophores fully corresponds to that characterizing the new host.

The rapidity with which these changes take place would appear to establish the fact that the expanded physiological state of these cells in albinos is produced by the direct action of a hormonal substance and not by influences mediated through the nervous system inasmuch as nervous connections are completely severed, and it would be difficult to conceive of their reëstablishment by the time these changes are manifested.

46 (1421)

## On the effects of ablation of the epithelial hypophysis on the other endocrine glands.

By **P. E. SMITH** (by invitation).

[*From the Anatomical Laboratory, University of California.*]

When the epithelial hypophysis is ablated in early embryonic stages in the frog, the resulting larvæ suffer in a characterisic way from defects in their pigment system. An equally definite set of alterations is produced in the other glands of internal secretion. Both Allen and the writer have reported the underdevelopment of the thyroid gland to which may in turn be attributed the failure of metamorphosis in these larvæ. The posterior lobe of the hypo-

physis in these larvæ is always present, though greatly underdeveloped—ample proof apparently of the need of coassociation with the epithelial portion of the gland. Most emphatic is the effect produced on the adrenal, whose cortical or interrenal substance is greatly decreased. This discovery was greatly facilitated by the employment of those methods which fix and stain the lipoids of the cortical tissue. These changes in the adrenal tissue do not occur in thyroidectomized larvæ and are consequently not to be referred to the thyroid reduction which is coincident with them.

47 (1422)

## On the occurrence of degenerative changes in the liver in animals intoxicated by mercuric chloride and by uranium nitrate.[1]

By WILLIAM DEB. MACNIDER.

[*From the Laboratory of Pharmacology University of North Carolina, Chapel Hill.*]

The following observations are based on the study of fifty-two intoxications by mercuric chloride and eighty-four intoxications by uranium nitrate. Dogs were employed for the experiments. In the animals intoxicated by mercuric chloride, the poison was administered by stomach tube in the dose of 15 mgs. per kilogram. In the uranium intoxications, the poison was given subcutaneously in doses varying from 4 to 6.4 mgs. per kilogram.

The experiments were terminated at different periods during the intoxication without employing an anesthetic. Such a termination has eliminated the acute degenerative changes in the liver which may develop very rapidly from the use of such an agent. The changes in the liver in both types of intoxications have shown great variation in their severity and the rapidity with which they occur.

### MERCURIC CHLORIDE INTOXICATIONS

All of the animals in this group, with eight exceptions, developed a severe gastroenteritis. The stools were frequent and

[1] Aided by a grant from The Rockefeller Institute for Medical Research.

contained blood and mucus. Twelve of the animals not only recovered from the gastroenteritis, but they failed to develop any delayed evidence of an intoxication. The remaining animals were either killed during the period of acute corrosive poisoning, or after having successfully passed through this stage, the experiments were terminated at different periods when the animals were suffering from the remote toxic effect of the poison.

As a result of these studies, the following observations have been made:

1. There is no relationship between the severity of the gastroenteritis and the extent of the degenerative changes in the liver. The degenerative changes in the liver consist first, in a deposition of fat in the liver cells surrounding the central vein of the lobule. The severer changes which follow consist in cloudy swelling and necrosis of these cells, and an extension of the process to the periphery of the lobule. The invasion of the necrotic area by endothelial leucocytes is usually not a prominent reaction.

2. The more extensive liver degenerations have occurred in those animals that have recovered from the acute gastroenteritis but have later shown remote evidence of the intoxication by the development of an acute kidney injury.

3. A final group of animals has recovered from both the gastroenteritis and the kidney injury, but at a later period has shown the gradual or rapid development of an acid intoxication and a kidney injury of sufficient severity to induce an anuria. The pathology of the liver in this group of animals has shown two types of response. Evidence of repair has consisted in finding liver cells with mitotic figures and occasionally large cells with more than one nucleus. Connective tissue cells are more numerous than in normal liver tissue. In addition to these changes of a chronic character that indicate the repair of some previous injury, the liver has shown acute degenerative changes which are most marked in the midzone and periphery of the lobule. These changes have consisted in an acute necrosis which is preceded by fatty infiltration and edema. In the areas of necrosis, the sinusoids are large and distended with blood.

## URANIUM NITRATE INTOXICATIONS

The earliest evidence of liver injury in uranium intoxications has consisted in the appearance in the liver cells of fat in the form of dust-like particles. This deposition is more marked in the cells immediately around the central vein than it is at the periphery of the lobule. Following this change, the cells show granular degeneration, an increase in size, and the deposition of fat in larger masses. The later changes have consisted in marked cloudy swelling, followed by edema and necrosis. Such a termination is more marked near the center of the lobule than at the periphery. As the cytoplasmic degeneration progresses, fat appears in the cells in large droplets, and extends to the periphery of the lobule.

The rapidity of the development, and the severity of these changes have shown no definite dependence upon the size of the dose of uranium employed in the intoxication.

The severity of the degenerative changes in the liver and the amount of stainable fat present in the liver cells have shown a relationship with the age of the animal in which the intoxication is produced. The older animals have shown a susceptibility to uranium intoxication which has been expressed by the more rapid development of a liver degeneration and by these changes being more extensive than has been the case with the younger animals.

Associated with the occurrence of the degenerative changes in the liver, the animals develop an acid intoxication. Such an intoxication is of a severer type in old animals than in young animals.

At present an investigation is in progress which is concerned with the relationship of the liver injury induced by both mercuric chloride and uranium nitrate with the development of an acid intoxication.

48 (1423)

## On the anti-spasmodic and anesthetic properties of benzaldehyde.

By DAVID I. MACHT.

[*From the Pharmacological Laboratory of the Johns Hopkins University, Baltimore, Md.*]

In several publications appearing elsewhere the author has described his investigations concerning the pharmacological properties of some benzyl esters on the one hand, and of benzyl alcohol on the other.[1]

Following these studies it was but logical to inquire into the properties of benzaldehyde, a chemical substance closely related to the above. Accordingly, experiments were instituted with the object of determining whether benzaldehyde exhibits the anti-spasmodic properties of benyzl benzoate on the one hand, and the local anesthetic properties of phenmethylol or benzyl alcohol on the other.

Benzaldehyde is sufficiently soluble in water (0.2 per cent.) to admit of experimentation on isolated tissues *in vitro*. Experiments with solutions of benzaldehyde on various isolated smooth-muscle organs were found to show that benzaldehyde relaxes the tonus and inhibits the contractions of such organs. Experiments with the drug on whole animals and observations of various organs *in situ* revealed also a sedative effect. Perhaps the chief exception to the rule was in case of blood pressure experiments. It was found that the pressure did not fall after injections of benzaldehyde solutions or suspensions except when large quantities were injected intravenously.

More interesting than the effect on smooth-muscle is the local anesthetic action of benzaldehyde. Experiments with aqueous solutions and more concentrated suspensions or emulsions of benzaldehyde showed that that substance possesses definite and marked local anesthetic properties. Thus it was found that it anesthetizes the sensory nerve endings of the frog's skin, of the

[1] *Journal of Pharmacology and Experimental Therapeutics*, 1918, Vol. 11, pp. 263, 389, 419.

cornea, and of the human mucous membranes. Furthermore, benzaldehyde solutions were found to paralyze also nerve conduction.

The toxicology of benzaldehyde has been worked out long ago, owing to its presence, in combination with hydrocyanic acid, in bitter almonds and other plants. As is well known, benzaldehyde is very little toxic, and can be taken by mouth in large quantities without any injurious effects. For this reason, it is official in the U. S. Pharmacopœia. The interesting local anesthetic properties of benzaldehyde found by the present author throw light upon the pharmacological action of compound tincture of benzoin and some other drugs. Practically, benzaldehyde is not as adaptable to clinical use as benzyl alcohol, because solutions of it are rapidly oxidized to benzoic acid. A detailed description of its pharmacological properties will appear in the *Journal of Pharmacology and Experimental Therapeutics*, and its relation to the therapeutic value of some well-known pharmaceutical preparations will be discussed more fully in a medical historical paper elsewhere.

49 (1424)

## A biological test for corpus luteum extracts in vitro.

By **D. I. MACHT** and **S. MATSUMOTO.**

[*From the Pharmacological Laboratory, Johns Hopkins University and The James Buchanan Brady Urological Institute.*]

The present authors have been engaged for some time in the study of the physiological action of various glandular extracts, and more particularly of their influence on the genito-urinary organs. In the course of these investigations, they have discovered a reaction produced by corpora lutea which it is deemed desirable to report in this place. It was found that aqueous or saline extracts of fresh and dessicated corpora lutea of various animals exert a powerfully stimulating action on the vas deferens and seminal vesicles. Small quantities of such extracts when introduced into a chamber containing a freshly excised vas deferens

preparation, suspended in warm oxygenated Locke or Tyrode solutions, stimulate the contractions of that organ, and the strength of the contractions is proportional to the strength of the drug introduced. All other glandular extracts tested, with the exception of the suprarenal and orchitic extracts, fail to elicit such contractions of the vas unless administered in very much larger doses. Epinephrin, the active principle of the suprarenal gland, stimulates these contractions more powerfully, while extracts of dessicated orchitic substance also stimulate these contractions only after doses twice as great as those of corpus luteum extracts.

The authors have studied extracts of fresh corpora lutea of the sow, and also extracts of various commercial preparations of the desiccated corpus luteum substance in respect to their action on the vasa deferentia of the dog, cat, rabbit, guinea pig, and the rat, and have found the most suitable and most sensitive preparation for testing the corporus luteum extracts to be a freshly excised vas deferens of the rat in Tyrode's solution. Such preparations, when treated with some corpus luteum extracts, may react by contractions in solutions corresponding to concentrations of 1:2,500 of the fresh gland, and they almost always react to concentrations of 1:1,000 of the fresh gland. It was interesting to note that the vas deferens, though very sensitive to the effects of the corpus luteum, does not react to extracts of ovarian substance proper. As far as the authors have been able to gather other data, both experimental and clinical, it seems that the activity of corpus luteum extracts, as indicated by the vas deferens preparations, runs parallel to the activity of those preparations as indicated by the other data. This organ, therefore, seems to furnish a convenient method of comparing the physiological activity of various corpus luteum preparations and some criterion for the testing of various chemical principles derived therefrom. Complete data of the present investigation will appear in due time in the *Journal of Urology*.

50 (1425)

## Arterial and venous oxygen in pneumonia and influenza.

By WILLIAM C. STADIE (by invitation).

[*From the Hospital of the Rockefeller Institute for Medical Research, New York City.*]

A technique for the puncture of the radial artery was devised which is simple and does no injury to the artery. The blood thus obtained was studied with respect to its oxygen content and the oxygen capacity by the Van Slyke gasometric method. In addition, venous blood was obtained by the technique of Lundsgaard and studied in the same way. Fifty observations were made upon twenty-five patients. Most of these cases had bronchopneumonia, usually post-influenza. A few had lobar pneumonia and some had uncomplicated influenza.

In normal controls the arterial blood varied from 85 to 98 per cent. saturated with oxygen. In patients with the type of respiratory diseases outlined above, the arterial blood is rarely more than 90 per cent. saturated. Some patients had as little as 85 per cent. of saturation without cyanosis, but, as a rule, when the arterial saturation falls below 85 per cent., it is associated with cyanosis. With an arterial saturation below 80 per cent. the cyanosis becomes marked, and in no case when the arterial saturation was below 80 per cent. did the patient recover.

In the influenzal type of bronchopneumonia the patient maintains his arterial saturation somewhere between 85 and 90 per cent. until twelve or twenty-four hours before death, when it falls rapidly. In one or two cases the arterial saturation was as little as 32 per cent., but this was six to twelve hours antemortem. These cases were all intensely cyanotic. In no case was there any striking diminution of oxygen capacity, even in the cases of marked septicaemia. In several cases where the low arterial oxygen saturation was associated with cyanosis, the disappearance or diminution of the cyanosis during recovery of the patient was accompanied by an increase of the arterial oxygen.

In general, the venous oxygen closely parallels the arterial in its per cent. of oxygen saturation, except in cases of failing circulation, where the venous oxygen is disproportionately low.

# SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

**Ninety-eighth meeting.**

*Schermerhorn Hall, Columbia University, March 19, 1919.*

*President Calkins in the Chair.*

51 (1426)

## The influenza epidemic of 1918 in the American Expeditionary Forces in France and England[1]

By **W. J. MACNEAL.**

[*From the Laboratory Division, Office of Chief Surgeon, A. E. F.*]

A disease, clinically recognized as influenza, became epidemic in the American Expeditionary Forces in France in May, 1918. Since August, 1918, the epidemic, previously mild, has assumed a more malignant character, often leading to a fatal broncho-pneumonia. In the fatal cases the lungs have presented a picture of malignant coalescing broncho-pneumonia frequently associated with hemorrhagic tracheo-bronchitis. The changes have varied considerably according to chronicity of the disease and the nature of the secondary infections. Influenza bacilli in large numbers have been found in the bronchi in fulminant cases. At most of the autopsies a mixture of bacteria was found in the respiratory tract, including pneumococci of various types, streptococci and sometimes staphylococci. Blood cultures during life were usually negative but showed pneumococci or streptococci in some cases.

Over work, exposure to cold and wet, inadequate nourishment, poor ventilation, inhalation of dust and general physical discomfort have diminished the natural resistance to the disease. The contagion spreads rapidly by distribution in the secretions of the nose and mouth, not only of the sick but of many other in-

[1] Publication authorized by the Surgeon General, U. S. Army. The full paper will be published elsewhere.

fected persons not suffering from the disease. The primary epidemic disease of the autumn is considered identical with that of the early summer, with the added complication of bronchopneumonia in the colder weather. The bacillus of Pfeiffer is the apparent cause of the epidemic disease but its causal relationship is not conclusively proved. Rest in bed, warmth and bodily comfort, promptly enforced at the outset, are the most important elements in the treatment. Prophylaxis includes avoidance of contagion and general hygienic measures to enhance natural resistance and retain it at a high level. Vaccines are of questionable value.

Influenza has been endemic in France for many years, and during the war this infection appears to have assumed a more virulent type in this country, small epidemics having been recognized in the British Army in the winter of 1916–17 and in the fall of 1917. American troops in France suffered very much from influenza, especially in the winter of 1917–18, the disease apparently being the same as that which became epidemic in 1918. The evidence suggests that the epidemic of influenza originated in France from the endemic influenza widely prevalent there. It is probable that the large numbers of American soldiers in France, subjected to strange environmental conditions, furnished a fertile soil for the propagation of the disease. The epidemic was evidently carried by ships from Europe to the United States and to South Africa.

52 (1427)

## The disinfectant action of glycerol in varying concentrations.

By C.-E. A. WINSLOW and DOROTHY F. HOLLAND.

[*From the Department of Public Health, Yale School of Medicine, New Haven, Conn.*]

Previous studies of the disinfectant action of glycerol have for the most part been conducted in relation to its supposed destructive action on invading organisms in vaccine virus. M. J. Rosenau made an exhaustive investigation of the subject in 1903 and concluded that "Glycerol has distinct but very feeble germicidal and

antiseptic properties." F. R. Blaxall reported a similar study in 1902–3, his work including only one concentration of glycerol, 50 per cent. More recently G. Mathers and G. H. Weaver have noted the remarkable viability (14 to 30 days) of various micrococci and streptococci in 50 per cent. glycerol suspension, and the persistence of the organisms for 90 days on blood agar lightly covered with 50 per cent. glycerol.

Our own work was undertaken in connection with the study of the specific effects of salt solutions upon the permeability of bacterial cells, and in connection with this investigation the effect of glycerol in concentrations varying from 9.2 per cent. to 100 per cent. has been determined, *Bacillus coli* being the test organism.

The glycerol used was an analyzed commercial preparation (Merck) with maximum limits of foreign substances .1814 per cent. The analysis showed absence of carbonizable matters, and heavy metals. Pure, ammonia free water was used in making solutions, and for the controls.

Glycerol solutions of varying concentrations (9.2, 27.6, 46.0, 64.4, 82.8, 100 per cent.) were made up in 50 and 100 c.c. portions. Young cultures of *B. coli* (16–20 hours) were washed from agar slants with 2–3 c.c. of the test solution. One c.c. of this suspension was added to the bottles of the solution, and after dilution,

VIABILITY OF COLON BACILLI IN GLYCEROL SOLUTIONS OF VARYING CONCENTRATION.

| Concentration of Glycerol. | Percentage of Number Originally Present Found After | | | | |
|---|---|---|---|---|---|
| | 2–3 Hours. | 4–5 Hours. | 5–7 Hours. | 8–9 Hours. | 18–24 Hours. |
| 0 | 99 | 108 | 114 | 110 | 146 |
| 9.2 per cent. | 82 | 118 | — | — | 185 |
| 27.6 per cent. | 117 | — | — | — | 52 |
| 46.0 per cent. | 87 | 77 | 55 | 50 | 17 |
| 64.4 per cent. | 75 | 41 | 18 | 10 | 5 |
| 82.8 per cent. | 53 | 38 | 15 | 6 | 4 |
| 100.0 per cent. | 8 | 4 | .6 | .35 | .005 |

agar plates were made. The solutions were incubated at 37°, one c.c. portions being removed at definite time intervals and plated after dilution. The test solutions up to 64.4 per cent. were shaken carefully by hand before removing the portion to be

plated; the solutions of high concentration, 64.4, 82.8, 100 per cent. were placed in a shaking machine and shaken for 5 minutes. Uniform distribution of the organisms was thus obtained even in the concentrated solutions.

The average results of 23 different series of tests are shown for typical time intervals in the table above. They suggest the following conclusions.

1. Glycerol in 9 per cent. solution exerts no appreciable effect upon the viability of *B. coli.*

2. Glycerol in solutions of strengths between 28 per cent. and 100 per cent. exerts a distinct disinfectant action, the effect increasing progressively with increase in the concentration, a 100 per cent. solution of glycerol causing the destruction of nine tenths of the bacteria present in three hours.

53 (1428)

## The effects of intravenous injections of dichlorethylsulphide in rabbits.

By ALWIN M. PAPPENHEIMER.

[*From Columbia University, New York City.*]

The effect of intravenous injections of dichlorethylsulphide (mustard gas) was studied in a small series of rabbits. The minimum lethal dose was found to be from 0.005 gm. to 0.01 gm. per kilo. The injection was followed by emaciation, diarrhea, and, in animals dying within a few hours following the injection, extreme restlessness, incoördinate movements, retraction of the head, and transient spasticity, but no definite paralyses or convulsions. Animals dying within twenty-four hours or so showed irregular pulmonary edema. The most interesting effects were found in the hemato-poietic system. Usually on the second day after the injection, the circulating blood showed a marked leukopenia, which in the terminal stages became extreme, leukocytes falling to 1,000 per cubic mm. or less. In animals which recovered there followed a gradual restoration to the original level. The leukopenia was accompanied by a relative but not absolute, mononucleosis. The erythrocytes appeared to be less severely injured.

A study of the bone marrow in these animals shows an effect comparable to that of benzol. There is early destruction of the cells of the granulocyte series and in some animals an extraordinary depletion of the bone marrow. Animals which partially recovered from the initial injection and were then killed, showed active regenerative changes in the bone marrow.

There is a possibility that these effects may have been due to the chlor-benzine or nitro-benzine used as a solvent in German gas shells, and carried over in the distillate used for injection. In view of the small amount of such impurity present, this does not seem likely, but there was no opportunity to repeat the experiments with dichlorethylsulphide completely freed from this solvent.

The experiments were carried out in collaboration with Capt. Morgan B. Vance, M.C., at Hanlon Field, Chaumont, France.

54 (1429)

## Developmental rate and the formation of embryonic structures.

By CHARLES R. STOCKARD.

[*From the Department of Anatomy, Cornell Medical School, New York City.*]

The eggs of different species develop at specifically characteristic rates. These rates vary for a given species within certain limits. Variation in the direction of increased rate is far more limited and more difficult to bring about experimentally than is the opposite variation towards a decreased or slower rate.

The slight acceleration of developmental rate that may be induced in early embryos does not seem to cause any marked deviation from the normal course of development, on the contrary such embryos are unusually well developed. It might be said that the ideal rate of development is probably somewhat faster than the so-called normal average usually followed. In other words, the normal conditions of development are not entirely the best possible conditions.

The limits of retardation in developmental rates are extremely wide. The rate may be slowed down to almost zero or development may actually be to all appearances stopped for long or short

periods of time in many species without noticeably injuring the embryo. Such an interruption normally occurs during the development of certain eggs as those of birds and some mammals. In eggs having a continuous development, such as those of fish, the rate of development may be slowed to apparent stoppage at many stages and held in such a condition for some time without injury to the embryo which results after the inhibiting influence has been removed. However, when the rate of development is retarded but not entirely stopped at certain critical periods and development is allowed to proceed at this diminished rate for some time, most serious structural anomalies are induced.

Double monsters of varying degrees of doubleness may actually be produced by slowing the rate at a time when the primary embryonic bud should arise. Normally the initial appearance of the primary embryonic bud probably suppresses the appearance of other buds which potentially exist, but when the primary bud is delayed in its appearance it becomes possible for more than one bud to arise, usually two. The distance apart of these two buds on the blastodisc determines the degree of doubleness of the resulting individual. Buds arising close together give two-headed monsters, while buds arising at opposite points on the periphery of the disk, 180° apart, each give rise to a complete individual, in such a case twins result.

When the two buds arise simultaneously they have equal chances in development and symmetrical double monsters result. If, however, one bud obtains the start over the other bud this start constitutes a supremacy which almost invariably makes it possible for the leading bud to develop into a perfectly normal specimen, and invariably defeats the possibility of normal development on the part of the slower bud.

An investigation of a large series of such double fish embryos lends strong support to the interpretation that the late bud is inhibited in its rate of development on account of the presence of the leading bud, just as the first bud to grow out from a notch on the leaf of *Bryophyllum* inhibits the growth of other buds as Loeb has so strikingly shown. The inhibited rate of development in the lesser component tends to suppress and interfere with the normal origin and development of certain organs, especially the

eyes and other head parts. Organs may entirely fail to arise, or develop abnormally after they do arise.

In the series of double fish when both individuals or both heads, as the case may be, are of equal size they are both normal, but whenever one component is larger than the other, the larger one is almost invariably normal and the smaller is *invariably* defective. This is not only true in the present series of specimens but also in all illustrations and descriptions of double monsters which I have been able to collect from the literature.

These embryos furnish material for an analysis of the causes of many common structural defects about which there has been considerable discussion, a consideration of this phase of the subject will be given in the complete review of the experiments.

55 (1430)

## A semi-lethal in Drosophila funebris that causes an excess of males.

By **O. L. Mohr** and **A. H. Sturtevant** (by invitation).

[*From the Zoölogical Laboratory of Columbia University, New York City.*]

In the course of the genetic work on *Drosophila melanogaster* cases have been found rather frequently in which the sex ratios showed marked deviations from the usual approximate equality. Those cases in which there is a deficiency of males have been the most frequent, and the explanation of many of these has been worked out by Rawls, Morgan, Bridges, Stark, and others. They are now known to be due to sex-linked lethal genes. Less frequently cases have been noted in which there was a deficiency of females (see Quackenbush, Science vol. 32). In none of these has the explanation hitherto been discovered.

From a culture of *Drosophila funebris* we obtained one female and 87 males. This female, mated to a few brothers, produced 60 females to 103 males. Descendants of this mating have been inbred for many generations, and have given sex ratios ranging from 0 ♀ : 76 ♂ up to approximate equality. There is no obvious

relation between the sex ratio of a culture and the pedigree of the parents—in fact, two cultures from the same parents may give quite different sex ratios.

Preliminary experiments indicate that environmental conditions—especially temperature—affect the sex ratio in this line; but we are not yet able to control it at will.

In this race the females frequently have abnormal abdominal bands; but this character appears in the males only very rarely. The evidence indicates that it is this character that is influenced by environmental conditions, and that the very abnormal females do not emerge from their puparia. Dark pupæ, evidently dead, are always to be found in cultures that give a significant excess of males. A few of these have been dissected, and have been found to contain dead flies with abnormal abdomens. In the few cases in which the sex was determined, these were females.

When the race here described is crossed to unrelated races, the sex ratio in $F_1$ approximates 1 : 1, and the $F_1$ females do not have abnormal abdomens. Both characters, however, reappear in the next generation. These crosses show also that the characters are both transmitted by males as well as by females.

These data indicate that abnormal abdomen is a recessive sex-limited mutation. It commonly affects only females, and the degree of the abnormality produced is dependent on environmental conditions. When the abnormality is extreme the females do not emerge, and an excess of males results.

56 (1431)

## The construction of chromosome maps.

By T. H. MORGAN and C. B. BRIDGES.

[*From the Zoölogical Laboratory, Columbia University, New York City.*]

The accuracy with which a chromosome map may be constructed depends upon several conditions. (1) The mutant characters employed should be carefully restricted to those cleanly separable both from the wild type and from each other, and whose viability is practically the same as that of the wild type. (2)

Mutants should be selected whose loci are properly spaced—not so close together that the error of random sampling is excessive, nor so far apart that double crossing over occurs between them. (3) When the amount of double crossing over between two distant loci is accurately known, data involving them can be used by making the appropriate correction. (4) The data must be obtained under uniform conditions, special attention being paid to the age of the parents, constancy and suitability of temperature, and to freedom from genetic modifiers of crossing over. (5) Any experiment involving more than two loci should figure only once in the calculation of each particular region of the chromosome. (6) Data for each region should be adequate in amount as judged by the laws of probability. (7) If slightly different positions are indicated by two or more independent experiments, then a mean position should be calculated in accordance with the amount and value of the different sets of data. (8) The framework of the map having been constructed on the basis of the most significant loci, each remaining locus is interpolated as accurately as the amount and reliability of data permit.

57 (1432)

## Effect of position of body on the length of systole and diastole and rate of pulse in man.

By WARREN P. LOMBARD and OTIS M. COPE.

[*From the Physiological Laboratory of the University of Michigan Medical School.*]

There is need of a practical method of determining the condition of the heart muscle in man. The contraction period of other muscles is lengthened if they are fatigued or degenerated, and this may be true of heart muscle. An accurate determination of the length of systole might be of use, provided its normal relationship to the heart rate and the ordinary variations were known.

At the Minneapolis meeting of the American Physiological Society December 28, 1917, the writers reported that they had studied the length of systole and diastole in man, by recording the carotid pulse and measuring the systole from the beginning of

the upstroke to the dicrotic notch. The subjects were 20 normal men, and 1,600 cycles were measured. A curve in which the average duration of systole and of diastole were plotted in relation to pulse gave a striking picture of the shortening of systole and diastole by increasing heart rate.

The great variation in the length of systole and diastole which may occur within a single minute was emphasized. Both are affected by respiration, and diastole, at least, by vaso-motor influences.

It can now be definitely stated that the changes in the length of the systole and the diastole observed in succeeding cycles have no constant relation to each other, and therefore are probably brought about in different ways.

The special object of this communication is to attract attention to the great difference in the average length of systoles and diastoles caused by a change in the position of the human body. It has been found that, in sitting the systoles average by pulse rates from 50–95.9 per cent. longer than in standing, and in lying down 17 per cent. longer than in standing. The diastoles are also lengthened, but only to about one half as much as the systoles.

The change in the length of the systoles caused by change of position of body, although of course influenced markedly by the pulse rate, is not due to the pulse rate alone, for the systole may be lengthened when the pulse rate has undergone no change, or when it is changed, the percentage change in systole may be much greater than that of the pulse rate.

58 (1433)

## The extraction of "fat-soluble vitamine" from green foods.

By THOMAS B. OSBORNE and LAFAYETTE B. MENDEL.

[*From the Laboratory of the Connecticut Agricultural Experiment Station, and the Sheffield Laboratory of Physiological Chemistry in Yale University, New Haven, Conn.*]

We have recently published experimental data to demonstrate the occurrence of "fat-soluble vitamine" in certain foods.[1] McCollum, Simmonds and Pitz[2] have stated that "ether extraction

[1] Osborne and Mendel, *J. Biol. Chem.*, 1919, XXXVII, 187.

[2] McCollum, Simmonds and Pitz, *Am. J. Physiol.*, 1916, XLI, 363.

of plant tissues does not remove the substances essential for growth which is contained in butter fat." They further say "owing to the large content of waxes, etc., extracted from plant leaves we have not been very successful in feeding ether extract from these sources." We have, however, obtained potent preparations as follows: Spinach leaves and young clover respectively, dried in a current of air at about 60°, were extracted with U. S. P. ether. The resultant green extract, yielding an oily residue equal to about 3 per cent. of the dried plant, was evaporated upon starch. These preparations, fed in daily quantities equivalent to 1–2 grams of the dried plant, promoted recovery and renewal of growth in rats declining in weight on diets deficient in fat-soluble vitamine. Inasmuch as only 30 milligrams per day of the ether extract of spinach sufficed for this purpose it appears that this product ranks among the most potent of the oils heretofore tested.

## ABSTRACTS OF THE COMMUNICATIONS, PACIFIC COAST BRANCH.

### Twenty-first meeting.

*San Francisco, California, March 5, 1919.*

59 (1434)

### Arsenic penetration of the meninges during the treatment of neurosyphilis.

By **H. G. MEHRTENS** and **C. G. MCARTHUR** (by invitation).

[*From the Stanford Medical School, San Francisco, California*].

It is difficult to estimate from clinical results the relative values of the different methods of treating neuro-syphilis. The amount of arsenic that reaches the cerebrospinal fluid may, however, be estimated quantitatively with reasonable accuracy, and the effectiveness of the treatment may be assumed to parallel this amount of penetration.

Quantitative estimations of arsenic penetrating the meninges was made in about 100 spinal fluids. These were divided into the following groups:

Group A—44 cases in which spinal drainage was performed one hour after simple intravenous injection of 0.6 arsphenamine.

Of this number 43 per cent. gave positive test for arsenic, averaging .036 m.mg. of arsenic per c.c.

Group B—23 cases in which the intravenous injection of 0.6 arsphenamine was followed in half an hour by complete drainage of spinal fluid. One hour later a second lumbar puncture was done to determine if complete drainage tended to increase the amount of arsenic penetrating. In 32 per cent. arsenic penetrated in half an hour; in 23 per cent. it had penetrated following drainage. Quantitatively, the average amount of penetrations in the first half hour was .009 and after one hour, .0043 m.mg. per c.c.

Group C—Of 5 cases in which complete drainage was done one hour before the intravenous injection of 0.6 arsphenamine none showed arsenic penetration an hour afterwards.

Group D—In 40 cases the patient's own serum was injected into the subarachnoid space followed in 6 to 8 hours by an intravenous injection of 0.6 arsphenamine. Of the spinal fluid obtained one hour later 92 per cent. showed positive test for arsenic. Quantitatively, these cases averaged .103 m.mg. per c.c.

From these figures it is apparent that the simple withdrawal of spinal fluid either before or after the intravenous administration of arsphenamine does not increase the amount of arsenic which penetrates into the spinal fluid.

On the other hand after the patient's own serum has been injected into his subarachnoid space, the injection of arsphenamine was followed by the appearance of arsenic in the cerebrospinal fluid in the great majority of the cases and the average amount of arsenic obtained was far greater than after simple arsphenamine injections. That the injection of serum caused meningeal irritation was showed by the subsequent cell count of 100 to 2,300 cells per cm. From these observations it may be surmised that the therapeutic results obtained in the Swift Ellis reaction depend in part, at least, upon a lowering of the barrier between the blood and spinal fluid owing to the meningeal irritation. This lowering allows arsenic freer access to the spinal fluid—a result which is in accord with Flexner's observations on the penetrations of antibodies.

*Conclusion.*—The intradural injection of serum 8 hours before the intravenous injection of arsphenamine definitely increases the penetration of arsenic into the cerebrospinal fluid.

# SCIENTIFIC PROCEEDINGS.

ABSTRACTS OF COMMUNICATIONS.

**Ninety-ninth meeting.**

*University and Bellevue Hospital Medical College.*

*President Calkins in the chair.*

60 (1435)

## The neuromotor system of Euplotes.

By **C. V. TAYLOR** (by invitation).[1]

[*From the Zoölogical Laboratory, University of California, Berkeley, Calif.*]

The fibrillar system of ciliate protozoöns was described by Sharp for *Diplodinium*, by Yocom for *Euplotes*. An integrated fibrillar system in the flagellate *Giardia* was described by Kofoid and Christiansen and for *Trichonympha* and *Trichomitus* by Kofoid and Swezy. The designation neuromotor apparatus has been given to this integrated organ system of the Protozoa, on the basis of morphological considerations and anatomical relations. Experimental proof of the conductile function of certain elements in this system has been accomplished in *Euplotes* by microdissection with the Barber apparatus. With this instrument actuating quartz microscalpels it has been possible to cut selected fibrils and determine the effects of the cutting and subsequent regeneration upon the previously analyzed stereotyped behavior of this organism.

*Euplotes patella* is a hypotrichous ciliate protozoön about 100–150 microns in length. Its neuromotor system consists of a small bilobed central mass, the motorium, at the corner of the cytostome. From this a fiber runs anteriorly and to the left at the base of the membranelles the full length of the adoral zone connecting with the bases of these motor organs. An extensive fibrillar

[1] Presented by Professor C. A. Kofoid.

lattice-work in the anteriorly projecting oral lip appears to be connected with this fiber. Running posteriorly from the motorium five distinct fibers diverge posteriorly, one each to the five posteriorly located anal cirri. No such fibers pass to the other thirteen cirri, but radiating fibrils from the base of each disappear in the adjacent cytoplasm. There is thus an integrated fibrillar system connecting the presumably sensory anterior lip, the powerful motor oral membranelles and the five major cirri of the ventral surface most active in the walking and swimming movements of the animal.

Structural relations do not indicate a supporting or skeletal function for these fibers. Observation of the fibers of living animals with moving cirri, and of the course and diameter of the fibers stained *intra vitam* and after fixation, showed not the least trace of contractility in any of the fibrillar components of the system except in cirri and membranelles.

The movements of *Euplotes* fall into two main categories, first creeping on the cirri, either (1) straight ahead, or (2) a quick backward movement, or (3) a turn aborally to the right; and second swimming movements of six types, (1) forward without spiral revolutions, (2) forward with spiral revolution, (3) circus movement to the right, (4) circus movement to the left, (5) sharp turn to the right, and (6) rapidly backward without revolutions.

Cutting experiments in which the anterior fiber or the anal fibers were severed were made to test the effect of cutting upon coördination, as revealed in these stereotyped modes of behavior. It is evident from transections of the body and excision of parts that the frontal cirri or anal cirri are indispensable to normal creeping movements, that the adoral membranelles are largely responsible for swimming movement (3), that the anal cirri function chiefly in the performance of creeping movement (2), and swimming movement (5), and that the adoral membranelles and anal cirri coöperate to effect swimming movement (6).

Cutting the membranelle fiber results in conspicuous differences in the behavior of the adoral membranelles on either side of the incision and in abnormal spiral revolutions in swimming.

Severing the fibers to the anal cirri affects both creeping and swimming. Creeping movement (2) is infrequent. Swimming

movement (5) was seldom observed and (6) was never seen after the fibers had been severed.

Destroying the motorium or cutting its attached fibers interrupts coördination in the movements of the adoral membranelles and anal cirri.

Any incision not severing either the membranelle fiber or the fibers to the anal cirri does not impair normal creeping or swimming movements although equal in extent to incisions which sever these fibers and result in loss of coördination. The conclusion is drawn that the fibrils of the neuromotor system are conductile.

61 (1436)

**The mechanism of boric acid hemolysis.**

By **M. KOSAKAI** (by invitation).

[*From the Department of Bacteriology, Cornell University Medical College, New York City.*]

Red blood corpuscles can be suspended, without direct injury, in a medium containing 1 per cent. of boric acid.

The corpuscles so treated are completely hemolyzed by sudden immersion in a suitable volume of isotonic saline or sugar solution, or in serum.

Sudden immersion of the treated corpuscles in any volume of concentrated saline or sugar solutions does not cause hemolysis and after such immersion the treated corpuscles are found to have lost their sensitiveness to immersion in isotonic saline solutions.

The gradual addition of a hemolytic volume of isotonic saline solution to the treated blood corpuscles causes no hemolysis and after such addition the treated corpuscles are found to have lost their sensitiveness to sudden immersion in isotonic saline solutions.

These observations indicate that the force operative in the "boric acid hemolysis" is that of osmotic pressure and this assumption is confirmed by the demonstration of a diffusion of the reagent into the corpuscles and by the fact that, for corpuscles that have been treated with a certain concentration of the boric acid, the minimal non-hemolytic concentrations of various substances are of identical "osmotic concentration".

62 (1437)

## The disinfection of vitalized tissues and the healing of wounds with chinosol and salt.[1]

By WILLIAM C. LUSK (by invitation).

[*From the University and Bellevue Hospital Medical College, N. Y. City.*]

The objective is to bring positive and convincing proofs of the healing value of chinosol in combination with salt.

Chinosol is oxyquinolin sulphate. *In vitro*, though a powerful antiseptic, it is very little germicidal. A 2 per cent. solution did not kill staphylococcus aureus in 24 hours. Its disinfectant action on vitalized tissues is therefore probably due to the excitement by it of physiological stimuli to bring nature's forces of resistance to the fore.

Salt was combined with chinosol by the writer through the influence of the writings of Col. Sir Almroth E. Wright[2] relating to salt in the treatment of wounds.

*Clinical experience.*—Examples of cases treated with chinosol and salt are as follows: Primary union in incised wounds, as for instance a case of cut tendons of the wrist; cases of acute suppuration, as one of cellulitis of the leg covering an area about the size of one's hand, due to colon bacillus, with sloughing interior, in which, with the use of a solution of 2 per cent. chinosol and 5 per cent. sodium chloride, the opposing surfaces of the abscess cavity were almost completely united on the ninth day; the healing of a whitlow with bone involvement without destruction of the tendons (function returning) and with union of the soft parts to the area of exposed bone, the latter having taken place by the tenth day, using a solution of 2 per cent. chinosol and 0.85 per cent. sodium chloride; the filling with granulations in about 5 weeks' time, of a bone cavity about 7 inches in length in an expanded lower portion of the shaft of a tibia, resulting from an operation for osteomyelitis, the whole medullary portion of the

[1] A foreword to an uncompleted paper read before the N. Y. Surgical Society, February 12, 1919.

[2] Wright, A. E., *Lancet*, 1915, II, p. 1009; 1916, I, p. 1203; 1918, I, p. 831.

## Addenda.

In dispensing the tincture of chinosol according to the formula here given, a heavy precipitate will form when the ingredients are first mixed with the alcohol, which on standing for 24 to 36 hours with occasional shakings of the mixture, will almost entirely dissolve. The precipitate consisting mostly of oxyquinolin, should be allowed to dissolve and should not be filtered off. When, in making up the tincture of chinosol, the chinosol and sodium chloride were first dissolved in the requisite amount of water needed to dilute the 95 per cent. alcohol down to 80 per cent., and the alcohol then added to this solution, the final residue after two or three days, was insignificant, which technic of making up this tincture seems to be the best. It requires 9.5 c.c. of water to be added to 45.5 c.c. of 95 per cent alcohol to make an 80 per cent. alcohol (U. S. Pharmacopeia), or 84 minims of water to 396 minims of 95 per cent. alcohol.

bone having been removed, treatment having been by a daily application of gauze wet with a solution of 2 per cent. chinosol and 0.6 per cent. sodium chloride for two hours; the healing of a pelvic fistula 6 to 8 inches in length, by injections, at first daily for one month with a solution of 2 per cent. chinosol and 5 per cent. sodium chloride, with which treatment the fistula became reduced to 2⅜ inches in length, later having been completely closed with the use of the tincture of chinosol; the cicatrization of a deep wound entirely encompassing the anus, the result of the separation of a slough, with high retraction of the anus above the skin surfaces of the buttocks, so that in 3½ weeks time the anus was pulled down and united even with the surrounding skin, treatment having been by the daily application of gauze wet with a solution of 2 per cent. chinosol and 2 per cent. sodium chloride for about ½ hour; the complete removal of a deep slough filling the base of a large carbuncle of the neck, which had been incised, with the adhesion of the undermined skin edges almost everywhere to the surface of the ulcer underlying them, by the fourteenth day, using a solution of 2 per cent. chinosol and 0.85 per cent. sodium chloride.

A chinosol ointment and a tincture of chinosol have important uses. The ointment (℞ chinosol grains vi, sodium chloride grains ii, lanolin and vaseline āā ℥ss) rubbed in for 4 or 5 minutes once in 2 or 3 hours, has proved a pretty reliable agent with which to abort beginning hair-follicle infections. The tincture (℞ chinosol 2 per cent. and sodium chloride grains i ss to the ounce in 80 per cent. alcohol) applied once a day to the skin around a furuncle, after having removed the grease with a fat-solvent, will prevent infection of neighboring hair follicles.

*The technic* is simple, application of the chinosol-salt aqueous solution in suppurating and granulating wounds which are accessible, being made by means of gauze which, when the wounds are discharging, is left in place between daily dressings, but when the wounds begin to granulate healthily with little discharge, should be removed in two or three hours following the dressing, to permit collapse of the wounds. The solutions used in this class of wounds contain 2 per cent. of chinosol with either 0.85 per cent. or 5 per cent. of sodium chloride. The combination of this strength of chinosol with the hypertonic salt probably promotes cicatrization

to a greater degree than does that with the isotonic salt, while the latter combination probably promotes the growth of granulations more than does the former. These solutions on contact with the wound cause a burning sensation which quickly passes away.

The healing of blind tracks of soft parts may be facilitated by injecting the tracks once in 6 or 8 hours through tubes having no punctures, introduced to their bottoms, for which purpose the 2 per cent. chinosol solution having the 5 per cent. salt content is probably the preferable solution of the two. The tincture of chinosol (℞ chinosol 2 per cent. and sodium chloride grains i ss to the ounce in 80 per cent. alcohol) has seemed particularly advantageous for the healing of blind fistulæ in ano, though with a different technic, it being injected into the track two or three times at 15 to 30 minute intervals each day.

For the control of sepsis in a draining empyema case, the solution of 2 per cent. chinosol with the 5 per cent. sodium chloride content is recommended, one ounce of which may be injected into the cavity to be retained by posture, following a preliminary washing with salt solution. In one case the use of a 5 per cent. salt solution for the preliminary washing, seemed to avail more toward cicatrization and healing of the wound than had normal saline. Wright has shown that a preliminary wash of a pus-secreting surface with physiological salt solution to remove the albuminous substances, gives an aftercoming antiseptic an opportunity to reach the bacteria. Before dressing a wound, the surrounding skin is first wiped with McDonald's solution (Alcohol 60 parts, acetone 40 parts, to which 2 per cent. of pyxol is added).

*First-aid treatment* is effected either by packing the wound with gauze saturated with a solution of chinosol iv grains to the ounce and 0.85 per cent. sodium chloride, which may be left in place for 24 hours before repairing or redressing the wound, when the same solution should be used again, or by simply sponging the wound freely with the solution during the operation for its immediate repair. The above gauze pack, after remaining in a fresh wound for 24 hours, often adheres to well nourished tissues. Hypertonic salt should not be used in a fresh wound which is to be sutured. In both old and fresh wounds which gape, it is advisable to interpose a piece of rubber tissue between the gauze which

brings the chinosol-salt solution into contact with the wound, and the external dressings, in order to prevent abstraction of the solution into the latter.

*Animal experimentation* was done to prove the value of chinosol as a first-aid disinfectant. In the animal experiments the wounds were constructed as pockets between the superficial and deep layers of the superficial fascia in a dog's back. These pockets, when made blood-free, would absorb the solution very freely no matter what was the strength of the salt, but when the tissues were infiltrated with blood the absorption of the solution would be slower or sometimes there would be none at all.

The instances in which primary union followed the disinfection of a scientifically infected wound, where the infection preceded the disinfection, were not frequent. One case which gave encouragement to the work, was that of a dog infected with staphylococcus aureus, having used as much of a 24-hour culture as could be taken up by a piece of gauze about 1 by 1¼ inches square, crumpled up, which was left in the wound for thirty minutes, the wound then being disinfected with a solution of chinosol grains vi to the ounce and 0.6 per cent sodium chloride, in which primary union took place in the disinfected wound, while from the control wound on the opposite side an extensive cellulitis developed, which resulted in a large area of superficial necrosis with ulceration extending from near the back bone forward to the anterior median line.

In a recent series of experiments performed on 12 dogs, in which the lymphatics leading from *wound pockets* between the layers of the superficial fascia, *uncontaminated with blood*, were first infiltrated with the disinfectant solution before infecting the wounds with as much of a virulent 24-hour culture of *Staphylococcus aureus* as could be absorbed on a piece of gauze about half an inch square, crumpled up and deposited in the bottom of the pocket for 30 minutes, and the disinfectant solution was applied to the wounds again following the infection, the wounds having been finally sutured primarily, these same wounds in seven of the animals united by primary union, while the controls all suppurated. The strengths of chinosol used in this series of animals were grains iv and vi to the ounce, and 2 per cent., and of sodium chloride, 0.85 and 0.6 per cent.

In two similar experiments with *blood-infiltrated wound pockets*, in which solutions of chinosol grs. vi to the ounce in combination with 0.6 per cent. and 0.85 per cent. sodium chloride respectively, were used, each of the wounds thus treated exhibited an area of dark gray staining of its fatty interior, due to a change produced in the infiltrated corpuscles in the course of from 15 to 30 minutes by the action of the chinosol, and both of the wounds suppurated, pure staphylococcus aureus having been found in the pus from each, while the control wounds also infiltrated with blood, both united by primary union. These results led to a study of the action *in vitro* of solutions of chinosol, and of chinosol and salt, on washed blood corpuscles. In this connection, it is of interest that two *sterile* blood-infiltrated wound pockets in the subcutaneous tissue of a dog, treated with 2 per cent. chinosol in combination with salt, with resulting areas of dark gray and gray-black staining, following primary suture, united by primary union. With the use of the first-aid solution (chinosol grs. iv = ℥ i and 0.85 per cent. sodium chloride), in experimental wounds into which blood had flowed, a smoky yellow color and occasionally a light grayish tinge have been noted, usually affecting the loose connective tissue joining together the superficial and deep layers of the superficial fascia, which at the same time has become the seat of an oedema resulting from an infiltration of it by the solution. In fresh traumatic wounds, staining of the tissues attendant upon the use of the first-aid solution, has, in a limited experience, not been a feature.

These experiments have shown that the production in fresh wound pockets uncontaminated with blood, of immunity to scientific infection with a large number of virulent *Staphylococci aurei*, by the use of chinosol with iso- and slightly hypo-tonic salt and once by the use of 2 per cent. chinosol alone, has been accomplished in a majority of the twelve instances in which it was attempted, which furnishes *proof* of the disinfectant action of chinosol on vitalized tissues. The practical application of this knowledge would be to the first-aid treatment of wounds. Thus it would seem that, if fresh traumatic wounds could, within the first few hours of their receipt, at a time when, as Carrel and Dehelly have shown, bacterial growth has hardly begun, have their

open lymphatics blocked with a solution of chinosol and isotonic salt, comparable to the lymphatic block with the disinfectant solution preceding the scientific infection with a large number of virulent bacteria in the dog's wounds, that immunity of these wounds, at least to the ordinary pus germs, in the presence of a but comparatively trivial amount of infection at this early period, could similarly be expected. The lymphatic block of a fresh traumatic wound with chinosol and salt should be superficial, since in the animal experiments an extensive infiltration of the solution into the lymphatics opening into a wound, following scientific infection of the latter, seemed many times to have been the probable cause of extending the infection to a distance from the site of its implantation. Although blood infiltration associated with the use of the chinosol-salt solution as described, might be incompatible with the production of immunity against a severe scientific infection in an experimental wound closed by primary suture, yet this same condition would not necessarily be incompatible with producing a lymphatic block against the invasion of the tissues by bacteria or with arresting bacterial growth, in a traumatic wound treated open by the introduction into it of gauze packing saturated with the disinfectant solution, especially when the latter is introduced early before the bacteria have begun to multiply greatly.

Twenty-four grains of chinosol in solution have been infiltrated into the lymphatics of a sterile, fresh wound in the back of a dog weighing 8 kilos, with primary union and without complication.

Miss W. Carey Noble, of the research laboratory of the New York Board of Health, has made very careful bacteriological tests in vitro with chinosol, which virtually confirm the tests of the Council on Pharmacy and Chemistry[1] of the American Medical Association.

Dr. Alexander O. Gettler, pathological chemist to Bellevue Hospital and to the City of New York, has done important work on the chemistry of chinosol to incorporate in this report.

Mr. Pro. V Prewitt, instructor in physiology at the New York University and Bellevue Hospital Medical College, has done a

[1] Report on Chinosol of Council on Pharmacy and Chemistry, American Medical Association, *Journ. A. M. A.*, 1910, LIV, p. 1801; editorial p. 1790.

valuable piece of work on the action of chinosol alone, and in combination with salt, on blood corpuscles.

*The merits of chinosol in combination with salt* as a tissue disinfectant can be summarized as follows: Its stability, its ease of application, its applicability to first-aid treatment of wounds, its tendency to dry up pus, its non-irritability when applied in accordance with the technic here advocated, unless possibly after prolonged use; also the facts that it appears not to attack tendons and that it facilitates the separation of sloughs.

The full scientific treatise on this subject will be published shortly in the *Annals of Surgery*.

63 (1438)

## The detection of small amounts of chloral in the presence of chloroform and formalin embalming fluid.

By ALEXANDER O. GETTLER.

[*From the Chemical Laboratory of the Pathological Department of Bellevue Hospital and that of the Chief Medical Examiner of The City of New York.*]

During the past five years the organs of many cases in which death was due to toxic substances, were submitted to complete chemical examination in this laboratory. Practically all of these were examples of sudden death occurring in the five boroughs of Greater New York for which the chief medical examiner or the medical assistant to the district attorney or both could find no anatomical cause. There were also a number of cases which were brought to my attention from other states. In this number, most of the commoner poisons, including chloroform, were represented. I have yet to encounter a case of straightforward chloral poisoning. Nevertheless, I became greatly interested in the question as to whether some of the so-called chloroform poisonings might not have actually been examples of chloral poisoning, and therefore, I made a study of various reactions to determine this point.

The several tests, namely, the isonitrile, the resorcin, the orcin

the alpha and beta naphthol, the Ragsky, the cyanide, the formic acid and the Vitalli Tornani, were studied, first, with relation to sensitiveness, second, as to the possibility of differentiating chloral from chloroform and, third, as to the interference of formalin with these reactions. The isonitrile and resorcin tests were found to be the most sensitive. By the former, chloral can be detected down to a dilution of .05 mgs. to the c.c. The resorcin test indicates chloral only to .25 mg. in one c.c., if the test is judged by the color alone; at this point the test is discarded if no red color appears. Chloral in amounts under .25 mg. per c.c. may still be present and detected by the appearance of a slight greenish fluorescence on diluting the reaction product with 10 c.c. of water. This fluorescence-giving property is sensitive to .05 mg. to the c.c. Both substances, namely, chloral and chloroform, respond equally well to the isonitrile test, the alpha and Beta naphthol, the Ragsky, the cyanide, the formic acid, the resorcin and the orcin tests. The reactions which are used for differentiating are as follows:

(*a*) The Vitalli Tornani test.[1] Its limitations are that it is sensitive only to the extent of 2 mg. in 100 c.c., that too much material must be used, and that it requires the use of a fairly large quantity of apparatus and, moreover, that all volatile halide compounds respond in a similar fashion.

(*b*) The Nessler test. This is given by chloral but not by chloroform and it is sensitive only to the extent of .25 mg. to the c.c. Furthermore, formalin gives a similar reaction and hence the test is useless when applied to embalmed material.

(*c*) The odor of chloroform in the distillate is possible, of course, only with large quantities of chloroform.

Formalin, which very often is present in the embalming fluid, interferes with the color reactions in that it likewise gives the same color, namely, a red or brownish-red.

The lethal dose of chloral is large, namely, 20 grams, but when one considers the instability of chloral, the rapidity with which the body detoxicates and excretes it, the time interval between the taking and the death of the individual (which is usually over 24 hours), the distribution in the various tissues and, finally, that

[1] Vitali Tornani, *Chim. Tossic.*, 1893, p. 179. Vitali e Tornani, *L'Orosi*, 1885, 7, 377.

steam distillation as ordinarily conducted in a general analysis recovers but a small fraction of the total amount present, it is obvious that only a few milligrams are to be expected. In several experiments I was able to recover, by steam distillation for a period of two hours, only 6 to 8 mg. from 100 mg. actually added.

In view of these facts, it is evident that the Vitalli Tornani test is to be used only when one is in a position to sacrifice practically all the material for this test alone. In a general unknown, however, this cannot, of course, be done, as a large part of the distillate is needed for testing the presence of various other volatile poisons.

The problem, then, resolves in finding a reagent which, first, is extremely sensitive to chloral, second, one for which only a small fraction of the suspected material need be used and, third, one which will react with chloral in a manner different from the reaction with chloroform and, fourth, if possible, one in which formalin will not interfere with the result.

A study of the following substances was made under different conditions of alkalinity (sodium hydroxide, sodium carbonate, calcium hydroxide).

Resorcin
Alpha naphthol
Beta napthhol
Orcin
Phloroglucin
Hydrochinon
Pyrocatechin
Phenol
Cresols
Thymol
Pyridine
Picric acid
Amino acids and boiling
Fatty oils and dehydrating agents
Yellow ammonium sulphide
Calcium sulph-hydrate
Sodium thiosulphate

The result reveals only two reactions which would serve the pur-

pose of differentiating chloral from chloroform and only one reaction in which formalin will not interfere.

The first reaction: phloroglucin and sodium carbonate with chloral, standing at room temperature for about one half hour gradually develops lilac to orange, to blood orange, to deep red. If extremely small quantities of chloral are present, the color develops to orange only, and if as low as .01 mg. in one c.c. is present, the color is a cross between orange and the light violet of the reagents themselves. Even in this extremely dilute solution a positive reaction may easily be recognized if compared with the control. Chloroform does not give this reaction. The color obtained with chloroform on the other hand is the slight violet of the reagents themselves. Formaldehyde and acetaldehyde interfere in that they give a reddish color. If the absence of aldehyde is shown by the reduced fuchsin test (which is not given by chloral), then the phloroglucin test may be looked upon as a sensitive method for purposes of differentiation. The technique is as follows:

To one c.c. of distillate add four drops of saturated phloroglucin and one c.c. of 20 per cent. sodium carbonate and allow to stand. Gradually the color develops, lilac to orange, to blood-orange, to deep red (sensitiveness, 0.01 mg. in one c.c.).

The reagents for the second method of differentiation are resorcin and sodium carbonate. Resorcin with sodium hydroxide and boiling have been used for a long time. Some workers, notably Schwartz,[1] the originator of the test, claim that besides the red color there is a green fluorescence given by both; others, notably Witthaus,[2] claim that only chloral gives the fluorescence. With the use of resorcin and sodium carbonate and at room temperature for one half hour I find, first, that with chloroform a green fluorescence never appears and that with chloral, even in minute amounts, fluorescence always occurs; second, that although the red color does not appear in very dilute solutions of chloral, the green fluorescence, especially if the reaction product is diluted with 10 c.c. of water, is always present; third, its sensitiveness is .01 mg. in one c.c.; fourth, formadehyde, acetaldehyde, formic acid, ben-

[1] *Ztschr. f. Anal. Chem.*, 1888, 27, 668.

[2] "Text-book of Medical Jurisprudence and Toxicology," Vol. 4, p. 1171.

zaldehyde, that is substances of an aldehyde character, do not give the reaction; fifth, if formadehyde is present together with the chloral, the reaction is not interfered with provided the test is done at room temperature. The technique follows:

To one c.c. of distillate are added 6 drops of saturated resorcin and one c.c. of saturated sodium carbonate solution or less if only traces of chloral are present. Let stand for one half hour, then dilute by adding 10 c.c. of water. A beautiful green fluorescence results. In cases of extremely small quantities of chloral, viewing by direct sunlight against a black, glossy background is of great advantage. Any development of color or fluorescence after many hours is of no consequence.

A series of 66 distillates from cases of suspected poisoning were examined by the above two described methods, with the following result:

| No. of Case and Organ Used. | Modified Resorcin Test. | Phloroglucin Test. |
|---|---|---|
| 19—Lungs | Negative | Negative |
| 20—Brain | " | " |
| 21—Brain | " | " |
| 23—Liver | " | " |
| 23—Stomach | " | " |
| 24—Liver | " | " |
| 25—Blood | " | " |
| 26—Uterus | " | " |
| 27—Brain | " | " |
| 28—Liver | " | " |
| 28—Stomach | " | " |
| 29—Brain | " | " |
| 30—Liver | " | " |
| 31—Brain | " | " |
| 31—Stomach | " | " |
| 32—Brain | " | " |
| 32—Liver | " | " |
| 33—Brain | " | " |
| 34—Brain | " | " |
| 35—Brain | " | " |
| 36—Liver | " | " |
| 37—Stomach | " | " |
| 37—Brain | " | " |
| 38—Stomach | " | "* |
| 40—Brain | " | "* |
| 42—Brain | " | " |
| 42—Stomach | " | " |
| 44—Stomach | " | "* |
| 44—Liver | " | " |

| | | |
|---|---|---|
| 45—Stomach | Negative | Negative |
| 45—Brain | " | " |
| 47—Liver | " | " |
| 51—Brain | " | " |
| 59—Stomach | " | " |
| 63—Brain | " | " |
| 64—Brain | " | " |
| 65—Brain | " | " |
| 65—Stomach | " | " |
| 73—Stomach | " | " |
| 74—Liver | " | " |
| 75—Stomach | " | " |
| 80—Lungs | " | " |
| 94—Stomach | " | " |
| 98—Stomach | " | " |
| 98—Intestines | " | " |
| 100—Brain | " | " |
| 101—Lungs | " | " |
| 102—Brain | " | " |
| 109—Stomach | " | " |
| 110—Liver | " | " |
| 113—Brain | " | " |
| 114—Liver | Slight red brown | Red brown* |
| 115—Liver | Negative | Negative |
| 116—Liver | " | " |
| 118—Brain | " | " |
| 119—Brain | " | " |
| 121—Liver | " | " |
| 123—Brain | " | " |
| 127—Brain | " | " |
| 130—Liver | " | " |
| 131—Liver | " | " |
| 132—Brain | " | " |
| 134—Liver | " | " |
| S/11—Liver | " | " |
| S/12—Brain | " | " * |
| S/13—Stomach | " | " |

Among the above collection most of the more common volatile poisons were represented. No. 80 and No. S/14 contained chloroform, Nos. 38, 40, 44, 114, S/11 and S/12 contained formaldehyde. None of them contained chloral.

The phloroglucin test is applied only if the reduced fuchsin test is negative. Six of the above set gave a reduced fuchsin test, due to the formalin of the embalming fluid. The remaining 60 distillates gave a negative phloroglucin test. All the 66 distil-

* Distillates from embalmed material.

lates gave negative resorcin tests. This shows that chloroform and volatile substances of the various organs will not respond to the tests.

Portions of brain, liver, kidney, lungs, stomach and intestine to which small amounts of chloral had been added (100 to 200 mg.) were distilled with steam. In each case both the modified resorcin test and the phloroglucin test responded strongly positive.

64 (1439).

**Immunity results from toxin-antitoxin injections.**

By WILLIAM H. PARK.

[*From the Department of Health of the City of New York.*]

It has already been reported that three injections of toxin-antitoxin given to children susceptible to diphtheria produced in about 90 per cent. sufficient antitoxin to give a negative Schick test and that the remaining 10 per cent. could be immunized by a second series of injections. Also that this immunity had lasted for 2¼ years.

It still remained to be determined whether this immunity would continue unabated, and also whether it is possible to immunize infants while immune from the antitoxin transmitted to them through their mothers. Tests have just been completed on several institutions in which 3¼ years have elapsed since the toxin-antitoxin injections. The immunity continues as well developed as in 2¼ years. It is hoped therefore, that the acquired immunity may persist possibly for life as in the case of natural immunity. Our ability to immunize the infants with passive immunity is being tested on a large scale, some 1800 infants having been injected. Only fifty of these have reached a period for testing. These show an immunity of 70 per cent. against an average immunity of those at the same age (8 months) not treated of 30 per cent. It seems therefore, that a very fair success can be achieved in young infants and that with greater knowledge it is possible to hope for a complete success. Small children and infants in contradistinction to adults show almost no local or general reaction to the injections.

65 (1440)

## Gastric response to extragastric irritation.

By W. HOWARD BARBER.

[*From the Department of Surgery, University and Bellevue Hospital Medical College.*]

Occasional experience with the contracting stomach in the open surgical abdomen has been confirmed and enlarged by similar experimental studies on the mammalian stomach. For this purpose, a dog is narcotized with morphia and ether, and opened, under surgical conditions, in the upper abdomen with the least possible mechanical trauma. The stomach is exposed and watched for contractions. If a typical animal, after the lapse of three minutes, two waves of peristaltic contractions occur on the exposed part of the stomach and follow each other at intervals of twenty seconds. With the stomach contracting in this manner, the gallbladder is seized in a crushing clamp for a few moments and released; similarly, the appendix or the duodenum may be clamped and released. In the great proportion of instances, there is a cessation of the stomach's motility for three minutes, more or less, followed by hypermotility after clamping in this manner the gallbladder, appendix, or duodenum. The experimental series is as follows:

TABLE SHOWING RELATION OF GASTRIC MOTILITY TO EXTRAGASTRIC IRRITATION.

| Exp. No. | Organ Irritated. | Change in Gastric Motility. |
|---|---|---|
| 10. | Duodenum | o |
| 11. | Parietal Periton | Inhibition |
| | Caecum (Appen) | Pylorospasm |
| | Gallbladder | Pylorospasm |
| | Pylorus | Retrostal.—Hypermotil.—Gastrospasm |
| 18. | Gallbladder | Hypermotil. |
| | Duodenum | o |
| 20. | Cecum (Appen) | Gastrospasm—Pylorospasm |
| | Gallbladder | Retrostal. |
| 22. | Gallbladder | Retrostal. |
| | Duodenum | Hypermotil. |
| 32. | Duodenum | Pylorospasm |
| | Gallbladder | o |
| 33. | Duodenum | Retrostal.—Pylorospasm |
| | Gallbladder | Retrostal.—Hypermotil. |
| | Parietal Periton | Inhibition |

| | | |
|---|---|---|
| 35. | Gallbladder | Hypermotil.—Pylorospasm |
| | Duodenum | Hypermotil.—Pylorospasm—Incisura |
| 40. | Parietal Periton | Inhibition |
| | Gallbladder | 0 |
| | Cecum (Appen) | Hypermotil. |
| 41. | Gallbladder | Hypermotil. |
| | Parietal Periton | Inhibition |
| 61. | Gallbladder | Hypermotil. |
| 68. | Cecum (Appen) | Hypermotil. |
| | Gallbladder | Hypermotil. |
| | Duodenum | Hypermotil. |
| 69. | Cecum (Appen) | Hypermotil. |
| | Gallbladder | Hypermotil. |
| | Duodenum | Hypermotil. |

In these thirteen experiments, there are eleven irritated gall. bladders, eight traumatized duodeni, and five crushed appendices, The respective gastric motor responses may be expressed in percentages as follows:

2. GASTRIC MOTOR RESPONSES IN PERCENTAGES.

| Organ Traumatized. | Hypermotil. | Hypomotil. | Retrostal. | Normal. |
|---|---|---|---|---|
| Gallbladder | 61.5 | 0 | 23.1 | 15.4 |
| Duodenum | 66.7 | 0 | 11.1 | 22.2 |
| Appendix | 100. | 0 | 0 | 0 |

After completing these experiments, the clinical records at Bellevue Hospital, Third Division, were reviewed from 1911 to the present time with the following result:

3. GASTRIC MOTOR RESPONSES IN PERCENTAGES (HUMAN).

| Organ Diseased. | Hypermotil. | Hypomotil. | Normal. |
|---|---|---|---|
| Gallbladder (19 cases) | 68.4 | 0 | 31.6 |
| Duodenum (8 cases) | 75. | 12.5 | 12.5 |
| Appendix (20 cases) | 55. | 0 | 45. |

66 (1441).

## The nature of osmotic pressure.

By **M. KOSAKAI** (by invitation).

[*From the Department of Bacteriology Cornell University Medical College, New York City.*]

The hemolytic effect of formaldehyde and that of urea, which were first observed by Eisenberg, are found to be, like that of boric acid, the result of osmotic pressure.

A comparative, quantitative study of the hemolytic action of these three substances shows that

(1) The same degree of osmotic hemolysis is not produced by identical "osmotic concentrations" of boric acid, formaldehyde and urea nor by a corresponding lowering in the concentration of the substances in the medium of suspension of the treated corpuscles.

(2) As the treating concentration of the three hemolytic substances is correspondingly diminished, the ratio between that concentration and the final concentration, in the hemolytic experiment, increases disproportionately with the different substances.

(3) The osmotic hemolysis of corpuscles which have been treated with the three hemolytic substances in the same "osmotic concentrations" is not inhibited by identical concentrations of sodium chloride nor of the hemolytic substances themselves.

All of these facts contradict the usual assumption that osmotic pressure is exerted directly by the solute.

These facts are easily compatible with the alternative view that osmotic pressure is merely the pressure of the water which diffuses through a semipermeable membrane to the side of higher "osmotic concentration," if, as is necessary, the factor of *time* is taken into consideration.

Under this conception, the degree of osmotic pressure developed depends not alone upon the original concentration of the solute but upon the length of time during which an effective difference in concentration is maintained on the two sides of the membrane. If the solutes are diffusible, as are all three of the hemolytic agents under study, the degree of osmotic pressure developed by them, under the conditions of the experiment, must depend, in part, on the *rate of diffusion* of the respective substance.

It is found that the diffusion rates of boric acid, formaldehyde and urea are respectively 90 seconds, 30 seconds and less than 5 seconds. These differences correspond with the differences in the hemolytic action of the three reagents mentioned above and they confirm the view that osmotic pressure is not a direct property of a solute but merely water pressure developed by the process of diffusion.

67 (1442).

## The results of the use of absorption of agglutinin on the identification of strains of influenza bacilli.

By WILLIAM H. PARK, ANNA W. WILLIAMS, and GEORGIA COOPER.

[*From the Department of Health of the City of New York.*]

The fact that Dr. Williams obtained abundant influenza bacilli in the pharynx of nearly all cases of influenza and that in the lungs of fatal cases she found them in pure cultures in about 20 per cent. of those taken, together with the similar results obtained in Boston and in some of the camps, strongly suggested the possibility that the influenza bacillus might be the causative agent in the epidemic. It seemed to us apparent that the different influenza bacilli from the different cases should necessarily belong to the same type if the influenza bacillus were the cause of the epidemic. We obtained, therefore, in pure culture strains from over 100 cases, and of these injected 20 individually into different rabbits. All of these strains produced a good quantity of agglutinin. In testing the strains with these twenty individual sera, we found the surprising fact that only four of the serums agglutinated any other strains beyond the ones used to immunize the rabbits. The technic of those doing this work was tested in every conceivable way without changing the results. Cultures were also passed through animals and grown on different media without altering these strains from the agglutination standpoint.

One of the investigators by accident received some of a fresh influenza culture into her throat. In forty-eight hours she developed an attack of bronchitis. The influenza bacilli found were identical in strain with those which were received from the culture. This holding of the strain characteristics in the secondary case is evidence that the strain does not quickly change. This evidence of multiple strains seems to us to be absolutely against the influenza bacilli isolated being the cause of the pandemic. It appears to us impossible that we should miss the epidemic strain in so many cases, while obtaining some other strain so abundantly. The influenza bacilli, like the streptococci and pneumococci, are in all probability merely very important secondary invaders.

68 (1443).

## A preliminary note on the experimental production of edema as related to "war dropsy".

By Emma A. Kohman (by invitation).

[*From the Hull Physiological Laboratory, The University of Chicago.*]

Denton and Kohman[1] report the occurrence of dropsy in a large per cent. of rats fed on a carrot diet with a low percentage of nitrogen. This dropsy is produced by a diet very similar to that of man in certain war zones where "war dropsy" has been reported.

It was of interest to determine whether the case of the edema was a lack of protein, rather than a deficiency of either of the vitamines, fat-soluble A, or water-soluble B, or a deficiency of salts in the diet.

The diets with which dropsy was produced in rats were made up of carrots, corn starch, fat (butter or lard), salts and an alcoholic extract of wheat germ. The percentage of all the ingredients, except carrots (the only source of protein) was varied, but in every case some of the rats developed marked edema, which manifested itself in various ways, sometimes with fluid in the pleural and peritoneal cavities, and sometimes with fluid collected subcutaneously, especially on the chest and about the fore legs. Weakness always occurred and usually loss of weight, except for a final gain due to the accumulation of fluid in the tissues.

In one experiment two out of three rats developed edema on a diet adequate in every way except for its protein content. In both cases the edema was subcutaneous about the neck and chest. One of the animals died. The other was fed a diet the same as the above described diet in every way, except that 18 per cent. of pure casein was added in place of 18 per cent. of corn starch. Marked improvement occurred in twenty-four hours and complete recovery in two days. After eating this diet for two weeks the animal was again given the original low protein diet but with double portions of salts. In two months the animal had again developed edema which was cured in the same way as above

[1] *J. of Biol. Chem.*, Vol. XXXVI, p. 249.

by substituting casein for corn starch, and in ten days had gained ten grams.

Another animal which had developed edema on the original low protein diet with the double salt mixture was cured with a diet the same in every respect except for the increased protein content.

That the low protein diet is adequate except for its protein content is shown by the fact that rats kept on this diet with 18 per cent. casein substituted for 18 per cent. corn starch grow normally, are active and in general good condition.

From the above observations and other experiments that are being conducted at the present time, it appears that "war dropsy" is not due to a vitamine deficiency as has been suggested by some, but is due to insufficient protein.

# SCIENTIFIC PROCEEDINGS.

## Abstracts of Communications.

### One hundredth meeting.

*Laboratory for Experimental Evolution, Cold Spring Harbor, N. Y.*
*May 24, 1919.*

*President Calkins in the chair.*

69 (1444)

### The results of selection with a Cladocera pure line (clone).

By **Arthur M. Banta.**

[*From the Station for Experimental Evolution,*
*Cold Spring Harbor, N. Y.*]

The writer has studied the effects of long-continued selection upon several parthenogenetic pure lines (clones) of three species of *Cladocera*, using their reactiveness to light as a basis for selection. In most of the lines the results, though suggestive, are inconclusive; or there is clearly no effect of selection; or (in two lines) the results even suggest slight differences in the reverse of an effect of selection. But with one line of *Simocephalus vetulus*, line 757, the result of selection is pronounced and convincing. This line was subjected to selection for a period of 54 months covering 181 generations of descent. In the final ten generations the strain selected for greater reactiveness to light had a reaction time less than one third as large as that for the strain of the same line selected for reduced reactiveness to light.

For the sake of obtaining averages showing less fluctuation than the average reaction times by single broods the data for each strain was averaged by two-month periods of the experiment (each period including the data for all the individuals tested in making the selections for six to eight generations).

For the first two-month period of the experiment the averages for the two strains coincide. But in the next period there is a divergence, the plus showing a greater reactiveness by a small margin. In successive two-month periods this divergence is in general increased. There are considerable fluctuations in the curves as are indicated by the facts,—that for two two-month periods, nineteen months after the experiment was begun, the minus strain was actually the more reactive of the two; and that during three other later two-month periods the mean for the minus strain approached that for the plus strain. But in general the means indicate increasing divergence in mean reaction time throughout the main portion of the experiment until for the last nine months the mean for the plus strain was just half that for the minus strain and during the last three months it was less than one third that for the minus strain.

It can be stated with assurance that with this material there is little, *if any*, relation between vigor and reactiveness to light and that in line 757 the effect of selection was not due to changes in the relative vigor of the two strains.

70 (1445)

**Exhibit showing the results of selection for a new Buff Race.**

By **C. B. DAVENPORT.**

[*From the Laboratory for Experimental Evolution, Cold Spring Harbor, N. Y.*]

A self-colored race of poultry was first produced in China about 1,500 years ago. All buff races of poultry have been derived from this Chinese race, the Buff Cochin. An attempt has been made to create a uniform buff race out of the Jungle Fowl. In 1907 a Jungle Fowl was crossed with a White Leghorn. This gave offspring in which the red of the Jungle Fowl appeared on a white ground, the black of the Jungle Fowl being hypostatic to the white. The birds showing largest amount of red were selected for breeding. During the first two years no progress was made but after that more highly colored red males and more

uniformly colored red females were produced. From 1909 to 1918 no evident improvement in the uniformity of the red coloring has appeared. The reason for the marked improvement in 1909 was due to the fact that, unintentionally, blood from the Dark Brahma (also an Asiatic breed) was introduced. This race has no Buff Cochin blood but a type of pattern which is more diffuse than that of the Jungle Fowl and hence permitted the buff pattern to break the limited bounds proscribed by its position on the Jungle Fowl.

Owing to the great amount of inbreeding which the experiment has necessitated and the increasing sterility and weakness of the offspring it will be necessary soon to bring the experiment to a close. The results obtained, however, speak strongly for the impotence of selection in an inbred line, with few genetical factors, unless new mutations afford new foundations upon which to advance. It is quite clear that the original Buff Cochin was not made by slow selection from a bird with the Jungle-fowl type of coloration. The Buff Cochin probably originated as a xanthic sport.

71 (1446)

## The influence of parental alcoholism upon habit formation in albino rats.

By **E. C. MACDOWELL** (by invitation).

[*From the Station for Experimental Evolution, Cold Spring Harbor, N. Y.*]

This report presents the training records of the children and grandchildren of a pair of rats heavily alcoholized daily for more than two months before the birth of the young. These alcoholized rats came from the same litter; another pair from this same litter was chosen on the basis of equal weight as the parents of the normal controls. All matings were between sibs in the same litter. Habit formation was tested by training on a Watson maze and the Yerkes multiple choice apparatus. The results of the maze training have been calculated on the basis of the last twelve trials of the training proper as well as twelve memory

trials given a month later. The criteria used are: average time per trial, number of "perfect" trials, number of wrong turns, or errors. Problems known as "first door to the left" and "first door to the right," with tests, were used on the multiple choice apparatus; the results are based upon the number of correct first choices, and the number of wrong choices.

It is obvious from the figures in the following tables that the rats that received alcohol and their unalcoholized descendants were less successful than the controls in meeting the situations presented.

## TABLE I.

MAZE PROBLEM.

| | Alcoholized. | Normal. | Parents. | | Grandparents. | |
|---|---|---|---|---|---|---|
| | | | Alcoholized. | Normal. | Alcoholized. | Normal. |
| *Training.* | | | | | | |
| Av. time per trial | 29.00 | 18.05 | 16.80 | 11.61 | 19.05 | 9.64 |
| Av. no. "perfect" trials | 1. | 4. | 1.25 | 4.33 | .41 | 5.50 |
| Av. no. errors | 2.56 | 1.44 | 2.47 | 1.01 | 2.84 | .62 |
| *Memory.* | | | | | | |
| Av. time per trial | ..... | ..... | 15.24 | 11.51 | 14.55 | 24.00* |
| Av. no. "perfect" trials | ..... | ..... | 2.75 | 5.66 | 2.16 | 5.41 |
| Av. no. errors | ..... | ..... | 1.78 | .91 | 1.71 | 1.19 |
| Number of rats | 3 | 3 | 6 | 6 | 5 | 5 |

* Due to one trial when one rat seemed sickly.

## TABLE II.

MULTIPLE CHOICE APPARATUS.

| Problem. | Correct First Choices. | | | |
|---|---|---|---|---|
| | Parents Alcoholized. | Normal. | Grandparents Alcoholized. | Normal. |
| I. | 4.21 | 4.91 | 3.88 | 6.12 |
| I. (test) | 5.75 | 6.05 | 5.58 | 6.70 |
| II. | 3.35 | 3.43 | 4.03 | 3.71 |
| II. (test) | 3.67 | 4.30 | 3.33 | 3.75 |
| | Wrong Choices. | | | |
| I. | 11.33 | 9.87 | 12.45 | 7.65 |
| I. (test) | 7.98 | 6.65 | 7.25 | 5.95 |
| II. | 17.32 | 15.53 | 14.47 | 13.05 |
| II. (test) | 13.97 | 10.67 | 13.83 | 13.00 |

72 (1447)

## Some factors influencing the human sex-ratio.

By **C. C. LITTLE** (by invitation).

[*From the Carnegie Institution of Washington, Cold Spring Harbor, N. Y.*]

In 1908 R. and M. De W. Pearl[1] published data derived from the vital statistics of the city of Buenos Ayres, concerning the sex ratio in the following types of matings: (1) Italian × Italian, (2) Spanish × Spanish, (3) Argentine × Argentine, (4) Italian ♂ × Argentine ♀, (5) Spanish ♂ × Argentine ♀.

Of these crosses it will be noted that three are not racial crosses and two are. The data obtained by the Pearls stretched over a period of ten years. Although including very large numbers their data has certain minor disadvantages, for the most part frankly recognized by the authors themselves. First in importance is the fact that their data do not take still births into consideration, and second, the Argentine race may properly be considered as itself somewhat of a mixture and therefore less likely to breed as a pure race. If however the first three crosses be recorded as pure racial matings and the last two as racial hybrids, the ratio of males to 100 females in the two types may be compared as follows:

TABLE I.

| | Total Cases. | ♂♂. | ♀♀. | Ratio. | Difference. | Diff. / P. E. |
|---|---|---|---|---|---|---|
| Pure matings ..... | 187,925 | 94,993 | 92,932 | 102.21 ± .16 | 3.78 ± .42 | 9.0 |
| Hybrid matings .... | 31,591 | 16,255 | 15,336 | 105.99 ± .39 | | |

It will be noted that there is a significant excess of males in the hybrids. The Pearls note this fact and state in addition that environmental differences also fail to account for the results. They further agree that experimental investigations are necessary in order to reach adequate explanations for the observed facts.

[1] *Biol. Bull.*, 15, 194–205.

It is believed that a close approximation to experimental conditions exists at the large lying-in hospitals. Here the patients are observed carefully, the sex of every offspring, including still births recorded, and the environmental conditions are for at least the period of confinement, as nearly equal as possible. The nationality of the parents of every child is recorded and the data thus obtained although not including nearly as large a number of cases as that of the Pearls, may, I believe, properly be considered accurate. During the past few months therefore, a study has been made of the records at the Sloane Lying-In Hospital, New York City. For this opportunity thanks are due to the staff and trustees of the hospital. The types of matings studied are those within each of the following races: English, Irish, Scotch, Italian, Russian, Greek, Austrian and German, and matings of all possible first generation types between these races. Matings of this latter category which produce racial hybrids are contrasted with matings within the pure races in the following table:

TABLE II.

| | Total Ind. | Males. | Females. | Ratio. | Difference. | Difference / P. E. |
|---|---|---|---|---|---|---|
| Pure stocks..... | 5,753 | 2,964 | 2,789 | 106.27 ± .91 | 15.29 ± 2.26 | 6.76 |
| Hybrids..... | 1,305 | 716 | 589 | 121.56 ± 2.06 | | |

It will be observed that although the numbers are smaller, the significance of the difference between the pure and hybrid stock is distinct. It will further be noted that both ratios are considerably higher than either of the Pearls.

In considering the probable effects upon their data of the missing still births the Pearls recognize the following three possibilities.

(*a*) That still births are distributed pro rata in pure and hybrid matings.

(*b*) That still births are more frequent in pure matings than in hybrid.

(*c*) That still births are more frequent in cross matings than in pure matings.

They recognize the fact that if either condition (*a*) or (*c*)

existed, their conclusions, based as they are on statistics leaving these births out of consideration, would be unaltered.

A tabulation of the percentage of still births occurring among the cases studied at the Sloane hospital gives the following result:

| | Total Cases. | No. of Still Births or Abortions. | Per Cent. of Still Births or Abortions. | Difference. | Difference / P. E. |
|---|---|---|---|---|---|
| Pure matings..... | 5,753 | 355 | 6.17 ± .21 | 2.19 ± .41 | 5.34 |
| Hybrid matings... | 1,305 | 52 | 3.98 ± .36 | | |

In the material studied, therefore, there appears to be a significant excess of still births in the pure matings as compared with the hybrid matings. Although the Pearls appear to consider this as a result unlikely to be obtained, it would seem that did still births depend upon a recessive factor or factors peculiar to certain races, there would be less chance for two gametes containing such factors to meet in a hybrid mating than in a mating within the race possessing the factor or factors in question.

Inasmuch as many embryos may reach birth although infected with syphilis it is likely that biological factors are involved in most if not all cases of still births resulting from syphilis. Such factors would determine susceptibility of the embryo in much the same manner as other biological factors produce strains or families with susceptibility to lung infections or some other weakness. It should be noted further that the results have proved significant in spite of certain rare unavoidable errors of classification, by which " hybrid " matings may be classed as " pure " and *vice versa*. Errors of this sort would tend to mask and not aid the results obtained.

The better record as regards still births is merely another way of stating that the $F_1$ racial hybrids in man would tend to show the vigor which has frequently been found to characterize racial hybrids in other mammals, such as rats, mice, and guinea-pigs. Such first generation hybrids would possess the factor complexes characterizing their respective races and would benefit by the summed up effects of all *dominant* factors of both races which might make for normal growth or development. That genetic factors which result in still birth are recessive, if they exist at

all, is obvious—for their lethal effect would naturally eliminate at once the individual possessing them were they dominant and their transmission to other generations would, therefore, be impossible. If we consider then the summation of characters for normal development and the probability that gametes containing recessive lethal factors will not be so apt to meet those of their own sort in a mating with an individual of another race, as in a pure mating, we have a possible explanation for the relative excess of still births in pure as compared with hybrid matings.

The ratio of sexes in the still births is as follows:

| | Male. | Female. | Uncertain. | Ratio. |
|---|---|---|---|---|
| Pure races..... | 175 | 161 | 9 | 108.69± 3.84 |
| Hybrids..... | 29 | 19 | 7 | 152.63±12.53 |

In both these cases when the ratios are compared with the ratios of Table II. it is found that the departures from the ratios there obtained are probably not significant. The difference between them being in the case of the hybrids 24 times its probable error and in the case of the pure matings only .6 times its probable error. It may, therefore, be concluded that in the material studied (1) a significant excess of males is observed in the progeny of human matings involving racial crosses as compared with matings within the race. (2) It may be predicted that racial crosses between the European races studied will produce in the first hybrid generation a significant excess of males. The economic importance of this principle to the United States is obvious, for our future population will largely be formed of such hybrids. (3) There are significantly fewer still births among the progeny of the hybrid matings studied as compared with the pure matings.

73 (1448)

## Sexual differentiation in the bread molds.

By **A. F. BLAKESLEE** (by invitation).

[*From the Laboratory of Experimental Evolution, Cold Spring Harbor, N. Y.*]

The Mucorales, represented by the bread molds, and their relatives, are characterized by a sharp sexual differentiation. Certain sexually primitive forms are hermaphroditic with equal gametes. From such forms as a possible starting point sexual differentiation seems to have proceeded in two directions—first toward a constant inequality in the size of the gametes seen in a few hermaphrodites; secondly toward a difference in the individual plants themselves. In these diecious forms, the interaction of two sexual races are necessary for the production of sexual spores.

Not only will the opposite sexes of a single species unite to form sexual spores but a reaction can also be induced between the opposite sexes of different species which shows itself as an "imperfect hybridization". An imperfect hybridization reaction between the sexual races of diecious species and heterogamic hermaphrodites leads one to believe that the (+) or vegetatively more active race is female and the (−) or less active race is male.

Tests of the sexual reaction of races of a large number of different species have been made and no race of the diecious forms has yet been found by the writer which reacts, if at all, other than as a male or a female. Variations, however, occur in the sexual vigor of different races and some are apparently neutral. Such a neutrality has been induced by environmental factors. Mutations have been observed in an hermaphroditic form producing hermaphroditic races with male, female and neutral tendencies.

74 (1449)

## The relation between the number of chromosomes of a species and the rate of elimination of mongrel blood by the pure-sire method.

By H. H. LAUGHLIN (by invitation).

[*From the Eugenics Record Office, Cold Spring Harbor, N. Y.*]

When to the neglect of individual Mendelian characters, the whole organism is taken into consideration, the common method of indicating the degree of parental qualities is to refer to the proportion of "blood" of one type or other which the particular offspring possesses. Thus the $F_1$ hybrid is called a "half-breed" or a "half-blood." Such an hybrid, bred back to the pure ancestor, gives a 3/4 blood pure, or a 1/4 blood mongrel, offspring, and so on according to the following schedule:

| | | | |
|---|---|---|---|
| $F_1$ | 1/2 blood. | $F_5$ | 1/32 blood. |
| $F_2$ | 1/4 " | $F_6$ | 1/64 " |
| $F_3$ | 1/8 " | $F_7$ | 1/128 " |
| $F_4$ | 1/16 " | | |

This nomenclature is used for all bisexual organisms. It is interesting to note that according to this scheme, theoretically there would always be a fraction of mongrel blood in each individual of a herd or group produced by the pure-sire method, regardless of the number of generations to which the process might have been carried. As a matter of practice, however, it is known that after a few generations the progeny of the pure-sire method are admitted on equal terms into the variety or species of the pure sire, and with no apparent injury to the purity of the paternal race. Thus Shorthorn breeders formerly permitted the registration of animals 31/32 pure.

According to Deniker, the male progeny of the $F_3$ generation of the mating of Spaniards of pure descent with Indians and part-bloods resulted in an individual who for all racial considerations was called a Spaniard and was taken into the social life of the Spanish people. In mating with the negro races the same consummation is achieved in the $F_4$ generation.[1]

[1] J. Deniker, "Races of Man," p. 542.

May mongrel blood be absolutely eliminated? If so, what are the chances that an $F_3$ offspring by the pure-sire method in man will carry absolutely no mongrel blood? The calculations are based upon the following assumptions: (1) that the haploid number of chromosomes in man is 12; (2) that all of the hereditary traits in the human species are carried by these 12 chromosomes, which, barring crossing over, non-disjunction and other special phenomena, segregate and recombine intact according to Mendelian expectation; (3) that the breeding between the pure sire and the mixed dams of a given chromosome-number in a given filial generation continues in proportion to the probable frequency of the latter's occurrence in each particular generation; (4) that all types of dams possess the same average fecundity.

*The chance that in man a given individual offspring produced by the continuation of the pure-sire method will carry absolutely no mongrel blood, i. e., will possess absolutely no chromosomes descended from the mongrel dam is measured by the following series:*

| | | | |
|---|---|---|---|
| $F_1$ | 0 to infinity | $F_6$ | 1 : 0.463 |
| $F_2$ | 1 : 4095.000 | $F_7$ | 1 : 0.208 |
| $F_3$ | 1 : 30.568 | $F_8$ | 1 : 0.100 |
| $F_4$ | 1 : 3.964 | $F_9$ | 1 : 0.049 |
| $F_5$ | 1 : 1.169 | $F_{10}$ | 1 : 0.025 |

*The chance that in man a given individual offspring produced by the continuation of the pure-sire method will carry not more than one (i. e., none or one out of a possible twelve) mongrel chromosome is measured by the following series:*

| | | | |
|---|---|---|---|
| $F_1$ | 1 to infinity | $F_6$ | 1 : 0.055 |
| $F_2$ | 1 : 314.077 | $F_7$ | 1 : 0.014 |
| $F_3$ | 1 : 5.313 | $F_8$ | 1 : 0.005 |
| $F_4$ | 1 : 0.829 | $F_9$ | 1 : 0.002 |
| $F_5$ | 1 : 0.205 | $F_{10}$ | 1 : 0.001 |

## SUMMARY.

1. Mongrel "blood" is eliminated by the pure-sire method not by "quartering out," but in segments, in degree governed by the relative ontogenetic potency of the particular chromosomes eliminated.

2. The degree of elimination of mongrel "blood" by the pure-sire method depends upon two factors, (*a*) the number of chromo-

somes characteristic of the species, (*b*) the proportion of mixed dams of each possible pure chromosome-number used in each generation, (*c*) the relative fecundity of dams of different pure chromosome-number, and (*d*) the number of generations through which the system is carried.

3. By the pure-sire method, without selection in the $F_5$ generation in a twelve-chromosome (haploid) species (including man), the probability that a given offspring carries absolutely no mongrel blood, i. e., mongrel-descended chromosomes is 1:0.205.

4. For mass improvement without selection the pure-sire method in a twelve-chromosome (haploid) species, ceases to be practically effective after the $F_5$ generation.

75 (1450)

## The transformation of the plant ovule into an ovary.

By J. ARTHUR HARRIS.

[*From the Station for Experimental Evolution, Carnegie Institution of Washington, Cold Spring Harbor, N. Y.*]

In plants there is a rather wide capacity for the development of organs of various kinds from primordia normally destined to produce quite different structures. For example, leaves may replace petals, stamens or carpels; petals may occur in the place of stamens or carpels. The transformation of stamens into carpels is a well-known phenomenon.

Furthermore, the continued development of a growing point the activity of which is usually terminated by the formation of some highly specialized organ, such as the flower or fruit, is quite familiar to those concerned with problems of variation.

Among these morphological abnormalities the continued meristematic activity of the axis which is normally terminated by the formation of the ovary is of very rare occurrence. It is, however, regularly found, although in a small and variable percentage of the cases, in one of the passion flowers, *Passiflora gracilis*. Here prolification of the fruit consists in the formation of series of carpels, which may or may not be ovuliferous, within the

normal fruit. The mass of accessory carpels thus formed may be so large as to rupture the fruit wall.

While the occurrence of prolification may be regarded as a heritable characteristic in *P. gracilis* the abnormality is of relatively rare occurrence. Physico-chemical factors must, therefore, determine the occurrence of prolification in certain fruits and its absence from others.[1]

If the formation of the basal prolification be due to the presence of special formative substances, one might occasionally expect to find the formation of carpellary tissue from other primordia. The only primordia normally developed subsequent to the carpels themselves are the ovules, which are borne on the carpellary margins.

To test this point, and to secure materials for other investigations, a series of dissections was begun in 1908. Those which were made from 1908 to 1915 are summarized in the accompanying table.

The results show that in the series of 568,098 dissections which have been made of fruits grown under a rather wide variety of conditions, basal prolification occurred 18,921 times, or in 3.330 per cent. of the fruits. Placental prolification occurred only 224 times or in .039 per cent. of the cases. Basal and placental prolification occurred in 18 of the 568,098 fruits.

While the occurrence of basal prolification presents a number of interesting morphological problems it does not seem to have the physiological significance of placental prolification. In the first case we have merely the continuation of activity of an axis which normally ceases with the laying down of the whorl of carpels forming the normal fruit. In the second case we have an entire transformation of a primordium. The primordium which should develop into an ovule forms instead a carpel, *i. e.*, one of the units of which the normal ovary is built up.

I am inclined to consider that this result is due to the local influence of special formative materials.

[1] A prolonged effort to demonstrate the nature of these factors has been inconclusive. Subsequent studies have not substantiated in all cases the position taken by Gortner and Harris (*Bull. Torr. Bot. Club*, 1913, XL., 27). Studies on the osmotic concentration and the electrical conductivity of the fluids of the proliferous mass and of the wall have been given by Harris, Gortner and Lawrence, *Biochem. Bull.*, 1915, iv., 52.

| Experiment. | Without Prolification. | Basal Prolification. | Placental Prolification. | Basal and Placental Prolification. | Total Placental Prolification. | Total Fruits. | Percentage Placental Prolification. |
|---|---|---|---|---|---|---|---|
| 1908 | 20,104 | 446 | .. | .. | .. | 20,550 | ... |
| 1909 | 116,821 | 4,622 | 30 | .. | 30 | 121,473 | .024 |
| 1911 | 30,105 | 873 | 9 | .. | 9 | 30,987 | .029 |
| 1912 | 10,487 | 441 | 1 | .. | 1 | 10,929 | .009 |
| 1913 | 123,216 | 4,458 | 17 | 3 | 20 | 127,694 | .015 |
| 1914 | 180,516 | 7,143 | 144 | 6 | 150 | 187,809 | .079 |
| 1915 | 67,686 | 938 | 23 | 9 | 32 | 68,656 | .046 |
| Total.. | 548,935 | 18,921 | 224 | 18 | 242 | 568,098 | .042 |

## 76 (1451)

## The antipyretic action of dextrose.

By HENRY G. BARBOUR.

[*From the Department of Pharmacology of the Yale University School of Medicine.*[1]]

Dextrose (30 gms. in 250 c.c. water) given per os with acetyl salicylic acid (1 gm.) in a case of chronic tuberculosis was followed within 2½ hours by a fall in rectal temperature from 38.03° C. to 36.02° C. In numerous observations upon this and other "labile" individuals under like conditions acetyl salicylic acid (1 gm. with 250 c.c.) never has produced a fall of temperature exceeding 1.1° C. in the same length of time.

### TABLE.

DEXTROSE GIVEN BY MOUTH. HUMAN SUBJECTS.

| Subject. | Date. | Dextrose, Gms. | $H_2O$, c.c. | A—S. A., Gms. | Rect. Temp. (° C.). | | |
|---|---|---|---|---|---|---|---|
| | | | | | When Given. | 2½ Hrs. Later. | Diff. |
| J. D. tbc..... | 4/22 | 30 | 250 | 1 | 38.0 | 36.0 | −2.0 |
| | 5/10 | 50 | 250 | 0 | 37.3 | 36.5 | −0.8 |
| | 5/14 | 75 | 250 | 0 | 37.7 | 36.8 | −0.9 |
| J. T. ac. art. rheum. ..... | 5/19 | 75 | 250 | 0 | 38.0 | 37.6 | −0.4 |
| M. M. D. normal. | 5/12 | 50 | 250 | 1 | 37.0 | 36.6 | −0.4 |
| | 5/15 | 100 | 250 | 0 | 37.1 | 37.1 | 0 |

[1] The expenses of this research are being defrayed from the Francis E. Loomis Research Fund of the Yale Medical School.

This observaton has led to an investigation of the action of dextrose upon the heat regulation.

The preceding table shows the effects of dextrose given per os upon the temperature of three individuals, one of whom was in normal health. (Readings were taken at half hourly intervals but the curves are uniform enough for the first and last reading to suffice.)

*Calorimetric Observations.*—In each of the above experiments also the heat production has been determined by the indirect method, using the Benedict Respiration Chamber at the New Haven Hospital. As would be expected, the $CO_2$ excretion was constantly above the basal levels for the same individuals; but since the temperature falls, it is evident that dextrose effects a marked increase in heat eliminating processes. The respiratory quotients were also high; but the metabolism data are as yet insufficient to make comparisons between febrile and normal individuals.

*Dextrose in Rabbits.*—The antipyretic action of dextrose given per os has been confirmed in five rabbits with "peptone fever." An average temperature fall of 0.8° in one half hour was obtained. The former level was usually regained in about two hours.

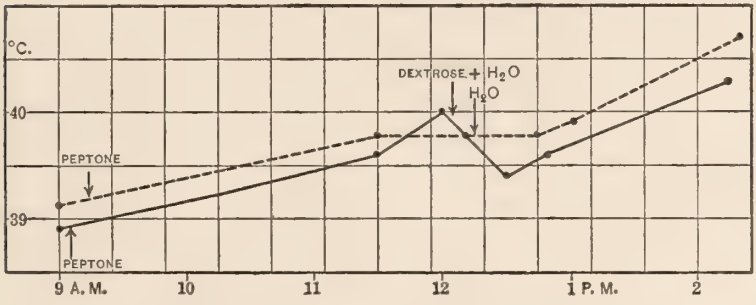



FIG. 1. Unbroken line: Temperature of 1,800-gm. rabbit. 9 c.c. of 20 per cent. "bactopeptone" (subcutaneously), followed in 3 hours by 18 gms. dextrose (by stomach tube) in 100 c.c. $H_2O$ at 41° C. Broken line: Temperature of 1,730-gm. rabbit. $8\frac{1}{2}$ c.c. of 20 per cent. "bactopeptone" followed in 3 hours by 100 c.c. $H_2O$ at 41° C.

The accompanying curve (Fig. 1) shows the temperature of an 1,800-gm. "peptone" rabbit given 18 gms. of dextrose by stomach tube in 100 c.c. of water heated to 41° C. The control

"peptone" rabbit (1,730 gms.) received only 100 c.c. of water (at 41° C.). The dextrose rabbit exhibited a loss of 0.6° C. in one half hour, the control rabbit no loss of temperature.

It is concluded that dextrose given by mouth under suitable conditions exhibits a decided antipyretic action, due to an increase in the heat elimination. The inference that the carbohydrate metabolism may play an important rôle in the action of antipyretic substances is being submitted to experimental inquiry.

77 (1452)

## Concerning the effect of prostate feeding on tadpoles.

By DAVID I. MACHT.

[*From the Johns Hopkins University, Baltimore, Md.*]

Gudernatsch was the first to call attention to the remarkable influence of the feeding of thyroid and thymus glands on frogs' larvæ. That observer noted that the thyroid causes a dwarfing or shrinkage of the growth and size of tadpoles, and at the same time very rapidly hastens their metamorphosis into frogs, while the thymus, on the other hand, causes giant tadpoles but inhibits their metamorphosis. Gudernatsch and other observers have also studied the effect of feeding of other organs and glands on the development of frogs' larvæ. As far as the present author has been able to ascertain, however, no experiments concerning the feeding of *prostate gland* to tadpoles are on record. The present author, in connection with a physiological and pharmacological study of prostatic extracts, conducted a series of experiments in feeding tadpoles with desiccated prostatic substance (Armour). The results are so interesting that it is deemed desirable to make a preliminary announcement on the subject in this place.

Prostate gland was fed to tadpoles of several species of frogs and it was noted that like the thyroid, the feeding of prostate substance tended to *hasten* the transformation of the larvæ into frogs. Such an effect was occasionally noted after administration of the gland substance for three or four days, and generally was distinctly noticeable after a period of from ten to fourteen days.

Unlike the effects of thyroid feeding, the feeding of prostatic substance while hastening metamorphosis did not produce much shrinkage in the size of the tadpoles. Indeed, it very often seemed to promote the growth of the tadpoles to a greater degree than was noted in the control animals. It was further noted, that prostatic substance was very much less toxic to the larvæ than was thyroid substance, so that the tadpoles could be fed on the prostate continuously without being killed. All kinds of control experiments with various glands and other tissues were made and, as a result of these, it was definitely established that the interesting effect on the metamorphosis of tadpoles was not produced by any other tissue except the thyroid and the prostate. A more extensive study on the feeding of prostatic substances of various animals to tadpoles, rats, and other animals is in progress and will be reported in due time in the *Journal of Urology*.

78 (1453)

## Non-protein sulphur of the blood: Its determination, its fractionation, and its clinical significance.

By MAX KAHN.

[*From the Department of Laboratories, Beth Israel Hospital.*]

Sodium citrate was used as anticoagulant. The protein of the plasma was precipitated by acetone—free methyl alcohol and zinc chloride (c.p.). The total sulphur was determined by oxidation with potassium chlorate and precipitation with barium chloride. The total sulphate was estimated by a method similar to the one of Vansteenberghe and Bauzil. The neutral sulphur was computed by subtracting the total sulphate from the total sulphur.

Studies were made on the blood of patients suffering with kidney, liver and malignant disease as well as those suffering with chronic infections.

79 (1454)

## Determination of carbon monoxide in blood.

By DONALD D. VAN SLYKE and HAROLD A. SALVESEN.

[*From the Hospital of The Rockefeller Institute for Medical Research, New York.*]

The blood is treated as in the determination of oxygen by Van Slyke's method.[1] A mixture of gases consisting of oxygen, carbon monoxide and the slight amount of nitrogen gas held in solution in the blood, is obtained. The extraction of the gases must be continued for a somewhat longer time than the one minute which is sufficient when oxygen alone is bound by the hemoglobin, otherwise the technique is the same. After the gases are measured, the oxygen is absorbed by permitting 1 or 2 c.c. of alkaline pyrogallol solution to flow slowly into the chamber of the apparatus from the cup at the top. The residual gas, corrected for the 0.009 c.c. of nitrogen gas per c.c. of blood known to be present, is the carbon monoxide.

80 (1455)

## Titration of organic acids in urine.

By DONALD D. VAN SLYKE and WALTER W. PALMER.

[*From the Hospital of The Rockefeller Institute for Medical Research, New York.*]

Carbonates and phosphates are removed by adding 2 grams of calcium hydroxide to 100 c.c. of urine, and filtering after 10 minutes. 25 c.c. of the filtrate is brought to a pH of approximately 8 by adding 0.2 N HCl with phenolphthalein as indicator, till the pink color disappears. Then 5 c.c. of 0.02 per cent. Tropeolin 00 solution are added, and the solution is titrated to a pH of 2.7 with 0.2 N HCl, the volume being brought to approximately 50 c.c. by addition of water towards the end of the titration. The color is compared with that of 50 c.c. of a control solution with

[1] *Jour. Biol. Chem.*, 1918, xxxiii., p. 127.

the same pH and indicator. The amount of 0.2 N HCl required to give the endpoint with a control in which water replaces the urine is subtracted. Of the organic acids known to be present in urine in quantitatively significant amounts, the titration measures from 93 to 100 per cent. of each. It also includes very weak bases, but apparently of this class of substances only creatine and creatinine are significant; they are titrated to nearly 100 per cent. The titration figure, corrected for the amounts of these two bases, represents the organic acids.

81 (1456)

## Some significant chemical changes in the blood coincident with malignant tumors.

By LUDWIG KAST and JOHN A. KILLIAN.

[*From the Department of Medicine and the Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital.*]

With the view of ascertaining the systemic effect of malignant neoplasms upon the organism, a series of sixty cases of various types of malignancies have been studied and contrasted with benign tumors. The data accumulated comprise determinations of the uric acid, urea, creatinine, sugar, diastatic activity and carbon dioxide combining power of the blood; the phthalein excretion; the occurrence of proteinuria and casts; and the blood pressure.

Two thirds of our cases of malignancies present evidence of an impairment of kidney function. The appearance and progress of this renal insufficiency follows the order characteristic of interstitial nephritis, described by Myers and his co-workers. The nitrogenous waste product first to be retained is uric acid, later urea and finally creatinine, and paralleling the accumulation of these nitrogenous substances there was noted a drop in the carbon dioxide combining power of the blood. A hyperglycemia and an increased diastatic activity pointing to a lowered carbohydrate tolerance, were encountered only in those cases mani-

festing a nitrogen retention, and as this retention became more marked, the hyperglycemia and increased diastatic activity kept pace with it. Apparently, then, the hyperglycemia and lowered carbohydrate tolerance, as gauged by the diastatic activity of the blood, must be attributed not specifically to the malignant new-growth but rather to the impairment of renal function. A further evidence of this impairment of kidney function was furnished by the decreased phthalein excretion, and the presence of protein and casts in the urine. Hypertension was noted in only a few instances.

This renal involvement was found to be associated invariably with general carcinomatosis; in 90 per cent. of carcinomata of the bladder, prostate, uterus and rectum and in about 50 per cent. of gastric carcinomata. On the other hand, carcinomata of the breast and epitheliomata produced no such changes. Of the small number of sarcomata coming under our observation, but one, a sarcoma of the kidney, disclosed a renal impairment. In all cases of non-malignant tumors no impairment of kidney function was noted. Further, the removal of the new-growth did not decrease the concentration of the nitrogenous substances in the blood, but in many instances, anesthesia and surgical procedures provoked an acute exacerbation of the condition. The termination of some of our cases was typically uremic. It is worthy of note that the chemical changes described bore no relation to the age of the patients.

Three cases of carcinoma of the pancreas have been studied by us. In two instances the diagnoses have been confirmed at autopsy. In all three cases, a hyperglycemia and a markedly increased diastatic activity were found, but in one instance only was there evidence of renal involvement. In this particular case there was an extreme asthenia and cachexia. All three cases showed glycosuria.

82 (1457)

## The toxic action of dichlorethylsulphide ("mustard gas").

By ALDRED SCOTT WARTHIN and CARL VERNON WELLER.

[*From the Pathological Laboratory, University of Michigan, Ann Arbor, Michigan.*]

The investigations recorded in these two papers are founded upon numerous series of animal experimentations and also clinical observations of human mustard gassing.

1. *Local Action: Skin.*—Dichlorethylsulphide ("mustard gas"), in liquid or in vapor form, even in very low concentrations, is an escharotic poison for the animal tissues (skin, conjunctivæ, cornea, mucous membranes of respiratory and gastrointestinal tracts) with which it comes in direct contact. The degree of the injury is proportionate to the concentration of the gas, the time of exposure, individual susceptibility, and local physical conditions, such as moisture, sweating, warmth, pressure and friction. The escharotic action is, for the greater part, painless, the anesthetic effect being especially notable upon the skin; while upon the mucous membranes its action may be more irritant, probably chiefly reflex in character. The cutaneous surfaces most susceptible are those with thinner, more delicate skin, well supplied with sweat glands and hair follicles, where sweat may collect, and which are exposed to friction or pressure, such as the axillæ, flexor surfaces, genitals, inner surface of arms and corresponding surface of trunk, inner surfaces of thighs, between the fingers, etc. There is a penetration of the gas into the sweat and sebaceous glands, and a re-solution of mustard gas vapor into the sweat or sebum occurs. The injuries are particularly striking in their insidious, slowly progressive development, becoming first apparent only some hours after the exposure. Upon human skin the lesion appears as a hyperemia, followed by vesication, eschar formation, sloughing and slow healing, with more or less pigmentation. Depilation may occur; in severe cases the eschar may extend entirely through the corium into the subcutaneous tissues. Secondary infection and gangrene of the eschars occur invariably

in cases not properly treated. Milder lesions may show only the earlier stages of hyperemia, vesication or pigmentation. In general the injuries may be classed as burns of first, second or third degree. Following extensive hyperemias in human skin a most marked pigmentation, exceeding in degree the most marked forms of solar tan may be quickly developed and fade slowly. This pigmentation may be diffuse or spotted. In human skin vesication is pronounced; in animals the cutaneous lesions are characterized by the development of marked subcutaneous edema in the injured area. The fluid of the vesicle or of the edema is nonirritating when applied to uninjured areas. In the case of human skin frequently exposed to very dilute concentrations (only perceptible by odor), an eczematous itching condition between the fingers, on the genitals, etc., may develop; rubbing or scratching of the itching part may lead to the quick development of a blister or superficial eschar (*Nikolsky's sign*). Such interdigital lesions in laboratory workers may resemble clinically those produced by the itch mite. Cutaneous areas injured by mustard gas are rendered more susceptible to trauma or other forms of injury, including new exposures to mustard gas. This local susceptibility is, however, a general one, and not a specific lowered resistance to the action of dichlorethylsulphide. Subcutaneous injections of pure dichlorethylsulphide produce painless eschars, followed by dry sloughing, with edema less marked than in the case of external cutaneous application; a hypostatic edema may develop on the animal's belly when injected in the back. In the tissues at the site of the injection and in the hypostatic edema mustard gas may be present for some days after the injection, as shown by odor and physiological reaction. The resolution of mild skin injuries is often attended by troublesome itching. Healing of the deep cutaneous eschars is very slow; during the healing of extensive deep lesions the patients complain of a sensation of tightness or contraction of the skin; large scars may be produced resembling those resulting from deep thermal burns. The hair may be lost; but when regenerated they may be white in color.

*Eye.*—Upon the cornea mustard gas exerts an especially injurious action, particularly at the vertex. Within ten to fifteen

minutes after exposure to dilute concentrations, degeneration or necrosis of the corneal surface may be demonstrated by the application of a 2 per cent. alkaline aqueous solution of fluorescein, the injured cells retaining a greenish fluorescent coloration. In more severe injuries the cornea may be killed throughout its entire thickness at the vertex. The mildest cases show a slight cloudiness; the severe cases present a characteristic porcelain appearance of bluish white opalescent cloudiness, often with a more opaque band or line running horizontally across the cornea just below its transverse diameter. The injury to the conjunctiva is shown by the development of a more or less severe catarrhal, seropurulent or purulent conjunctivitis with marked edema of the subconjunctival tissues leading often to "ruffling" of the lids, entropion, ectropion or a combination of these. Even the lighter cases tend to run a chronic course with disturbances and reduction of vision. In the severe cases cicatrization and vascularization of the cornea take place slowly with resulting impairment or loss of vision. The injured eye is more susceptible to infection; and in infected cases suppurative panophthalmitis may develop with complete destruction of the eyeball. Recovered cases of mild mustard gas conjunctivitis often show an increased sensitivity to the action of light, dust, and other irritants, including mustard gas fumes.

*Respiratory Tract.*—Upon the mucosa of the respiratory tract mustard gas vapor produces a local injury to the epithelium as shown by the development of a catarrhal, desquamative, membranous, diphtheritic or purulent inflammation (rhinitis, stomatitis, pharyngitis, laryngitis, tracheitis and bronchitis), these lesions being most severe in the nose, back of tongue, palate, pharynx and larynx, decreasing in intensity downwards. Coryza, salivation, dryness of mouth and throat, aphonia and persistent cough are the chief symptoms, with physical signs of laryngeal, tracheal and bronchial involvement, and atelectasis, emphysema and edema of the lungs. As a result of secondary infection a purulent bronchopneumonia may develop.

*Gastrointestinal Tract.*—Through the swallowing of air, saliva or secretions from the upper respiratory tract containing mustard gas, or from the ingestion of contaminated food local corrosive action upon the alimentary mucosa may be produced, varying

from a catarrhal inflammation to large areas of eschar formation with resulting gastric ulcer, perforation, etc. The symptomatology of the mildest lesions is covered up by that resulting from the more severe burns elsewhere; the more severe ones will produce marked symptoms referable to the stomach and intestines.

2. *General Action: Susceptibility.*—There exists a racial (whites more susceptible than negroes) and an individual susceptibility to the action of dichlorethylsulphide, particularly in the case of the skin and probably also of the respiratory tract. The individual susceptibility, in some cases at least, is associated with the characteristic stigmata and symptomatology of the thymicolymphatic constitution. Acquired susceptibility is not specific. Animals show also generic and individual differences in sensitivity to mustard gas.

*Systemic Action.*—There is no evidence of any systemic poisoning by the absorption of dichlorethylsulphide from the skin, eyes or mucous membranes of the respiratory or gastrointestinal tracts. There is no metastatic action of the gas from the site of local external application.

*Shock.*—In all severe cases of mustard gas burns of skin, eyes, or mucous membranes there is usually the clinical picture of severe shock, in the form of intense pallor, depression of pulse and temperature, general collapse, nausea and vomiting. The mildest cases show no systemic reaction.

*Blood and Urine.*—No changes are observable in the blood or urine of mild cases. In cases with large infected burns of skin or respiratory tract, the blood presents a mild secondary anemia with leucocytosis; we have never observed leucopenia; the blood urea is increased; the urine is diminished, concentrated, and contains casts and albumin. Under forced fluids the urinary symptoms improve, and the blood urea diminishes. In severe infected cases the general picture may be that of a severe toxemia.

*Intravenous and Subcutaneous Injection.*—When injected intravenously or subcutaneously dichlorethylsulphide is an active poison, causing death in one to four hours intravenously and two hours to three weeks after subcutaneous injections (for rabbits intravenous injections of .0075 c.c. per kilo may be lethal within four hours), according to size of dose, individual animal, etc.

When death takes place quickly, the symptoms are chiefly those of an action upon the central nervous system, such as hyperexcitability, rapid respirations, general convulsions, opisthotonos, gradual failure of respiration and circulation, coma and death. When the animal lives longer after small intravenous injections, or after subcutaneous injection, there develops a characteristic symptomatology of salivation, marked diarrhea, and fall of temperature, with marked anorexia, emaciation and depression. With subcutaneous injection of .015 to .06 c.c., death usually takes place from the fourth to the tenth day.

83 (1458)

## The pathology of dichlorethylsulphide (" mustard gas ") poisoning.

By ALDRED SCOTT WARTHIN and CARL VERNON WELLER.

[*Pathological Laboratory, University of Michigan, Ann Arbor, Michigan.*]

The specific microscopic pathology of the local lesions of dichlorethylsulphide poisoning consists in degeneration and necrosis of the cells with which it comes in contact. The earliest microscopic change is pyknosis of the nucleus and cell body, followed by hydropic degeneration, liquefaction or coagulation necrosis. In the skin, hyperemia, with regeneration of the damaged cells, pigmentation, vesicle formation, desquamation of the dead epidermis or eschar formation mark varying stages of severity of the lesion. The degenerative changes extend deepest in the hair follicles and sweat glands. In mild burns without vesication the papillary layer of the corium may show a greater degree of necrosis than the epidermis itself, thus explaining the frequent occurrence of Nikolsky's sign. Large pigmented chromatophores may be the only living cells left in the papillary layer. In severe burns the necrosis may extend entirely through the corium. In the cornea, pyknosis and simple or coagulation necrosis of the corneal epithelium and interstitial substance, even to the endothelial layer, in extent varying with the degree of

exposure, constitute the microscopic features. On the conjunctivæ the epithelium shows pyknosis, hydropic degeneration, liquefaction necrosis, or there may be a deeper necrosis extending into the subconjunctival tissues. The conjunctival surface suffers to a less degree proportionately than the epidermis. On the mucous membranes the epithelium shows pyknosis, hydropic or mucoid degeneration, desquamation, liquefaction or coagulation necrosis. The necrosis may extend into the submucosa, but the depth of the lesions on the conjunctivæ and the mucous membranes of the respiratory tract is never so great from identical exposures, as it is in the skin. Following the necrosis there is marked hyperemia, and the development of an edema, more marked in the subcutaneous and subconjunctival tissues in animals, but less marked in man. Human skin, however, shows a much greater tendency to vesication. The blood vessels in the necrotic area are killed, the blood cells hemolyzed to some extent without thrombus formation or much extravasation, except minute hemorrhages by diapedesis. Following the lesion there is a demarcating inflammation, with slow regeneration, repair or cicatrization. The regeneration of the epidermis proceeds from the epithelium of the sweat and sebaceous glands. On the mucous membranes there results in the severe cases a localized eschar or ulcer, or a more diffuse diphtheritis. With secondary infection the inflammatory process becomes purulent or suppurative. The influence of secondary trauma and infection is well shown in the early development of deep areas of decubitus in the injured regions of the skin. Multiple furuncles may develop, or large cutaneous areas become gangrenous. In the eye purulent involvement of the anterior chamber, iris and ciliary body may occur, or even a suppurative panophthalmitis. In the respiratory tract secondary infection of the injured mucosa may lead to a purulent bronchopneumonia.

The internal organs in animals with mustard gas lesions of the skin, eyes, respiratory or gastrointestinal tract offer nothing of a specific pathologic nature. There is general congestion, marked splanchnic congestion, acute catarrh of the intestines and, in infected cases, some cloudy swelling of the kidneys.

In fatal cases the cause of death is to be found in shock,

secondary infection with toxemia, or local conditions as laryngitis, tracheitis, bronchitis and bronchopneumonia. It is also possible that the entrance into the body of shell fragments carrying liquid dichlorethylsulphide might cause a relatively speedy death through absorption.

At the site of subcutaneous injections there is found a local eschar with demarcating hemorrhage, edema and inflammatory infiltration; in the large veins into which injections have been made, no changes have been found except occasional thrombosis.

The general pathology of the injected cases presents a specific pathologic picture in the intestinal tract in the form of a severe mucoid, desquamative or necrotic enteritis, the intestinal epithelium showing the most marked hydropic or mucoid degeneration, even to liquefaction necrosis. Similar changes may be found in the epithelium of the bile ducts. In a certain number of cases the spleen, lymph nodes and hemal nodes show a marked hemosiderosis, the hemosiderin being contained in large hemophages. It is most probable that these evidences of increased hemolysis are explainable by the extravasations and blood destruction occurring at the site of the injection. No specific changes were observed in the blood-forming organs. In the other organs no pathologic changes but congestion have been found, with the rare exception of emboli or thrombi.

*Mode of Action.*—The cause of death in intravenous and subcutaneous injections would appear to be the direct action of minute quantities of free dichlorethylsulphide or some poisonous product resulting from its decomposition, upon the cells of the central nervous system. It has been assumed that the pathologic action of dichlorethylsulphide is due to its hydrolysis within the tissue cells. The products of this hydrolysis, hydrochloric acid and dihydroxyethylsulphide, when injected into the blood, do not produce the same effects. Dihydroxyethylsulphide and hydrochloric acid, when injected into the circulation in much larger doses than would result from the hydrolysis of the fatal doses of mustard gas, are harmless. The effect upon the cells of the central nervous system may, however, depend upon hydrolysis, with the liberation of hydrochloric acid (*Marshall*), in these cells of minute quantities of mustard gas in the circulation, or these

cells may be injured without such hydrolysis occurring. It is probable that the gastrointestinal catarrh resulting from the injections of dichlorethylsulphide is secondary to the nervous injury, rather than to an excretion of the poison or poisonous products through this tract, although this point remains unsettled. No positive tests for dichlorethylsulphide or dihydroxyethylsulphide have been obtained in the bile, intestinal contents, or urine. Incidentally, it has been shown that the platinic chloride-sodium iodide color test for dichlorethylsulphide is not applicable to the body fluids or extracts of various organs and tissues, as similar color changes are produced by some of these.

*Treatment.*—The principles of treatment to be applied to mustard gas injuries are primarily those that will remove any of the gas remaining, lessen necrosis, prevent infection and promote healing. Our experience leads us to recommend the use of Dakin's solution in irrigation or full baths for the skin lesions, and a 0.5 per cent. solution of dichloramine-T for the eye lesions, and also as a mouth wash. The fluid intake should be forced when the urine is concentrated. Pressure must be removed from all injured areas. Air-excluding and infection-including protections, such as oily dressings, paraffin sprays and coatings, zinc stearate, olive oil, vaseline, etc., should not be used, unless there is an active and persistent germicidal agent present as in the case of sodium stearate impregnated with chloramine-T, or the chlorcosane solution of dichloramine-T.

*Sequelæ.*—Among the most important sequelæ of mustard gassing is the apparent increased susceptibility to influenza, bronchitis, pneumonia and tuberculosis following lesions of the respiratory tract cicatricial contractions, pulmonary fibrosis. The respiratory infections may become chronic and death from these may take place months after the gassing. Persistent aphonia, due to local lesions or as one expression of a traumatic neurosis, is not a rare sequel. Chronic disturbances of vision are also in part the result of local changes and in part psychical. In the skin conditions of chronic eczema, itching and desquamative dermatitis, and pigmentation occur as sequelæ. Leucotrichia has also been observed. It is safe to predict that a development of squamous cell carcinoma in the extensive cicatrices, following mustard gas

lesions will take place fifteen to twenty-five years later as in the case of extensive thermal burns. Finally, the psychical disturbances following mustard gassing should not be minimized.

As to its use in warfare, mustard gas is a disabling rather than a killing agent. Under the actual conditions of the field the great majority of mustard gas casualties are likely to be of a nature tending to incapacitate the injured for service for a number of days or weeks, or even for months. Added to this, the insidious character of this invisible fire, painless and often unrecognized in its action, makes mustard gas a potent factor in undermining the morale of the troops exposed to it.

## Abstracts of the Communications, Pacific Coast Branch.

### Twenty-second meeting.

*Berkeley, California, May 15, 1919.*

84 (1459)

### The optimum H-ion concentration for the growth of B. typhosus, and the effect of changes in H-ion concentration on the generation time.

By **P. Schoenholz** and **K. F. Meyer.**

[*From the George Williams Hooper Foundation for Medical Research, University of California Medical School, San Francisco.*]

1. *B. typhosus* has a range of growth from $P_H$+ 5.0 — $P_H$+ 8.6, with an optimum growth at $P_H$+ 6.8 — $P_H$+ 7.0.

2. Stock cultures isolated from stools, blood, and urine have a more decided optimum than recently isolated cultures. In such cultures, the plateau is much more pronounced and extends over a wider range than in stock cultures. The latter is suggestive of microbic adaptation to changes in H-ion concentration in body fluids, particularly urine and bile.

3. The growth curve is influenced by changes in H-ion concentration.

At $P_H$+ 7.0, the minimum generation time at 36.0° C. — 37.0° C., is 31 minutes. Maximum growth begins on the average after five hours incubation.

At $P_H$+ 7.8, the minimum generation time is 33.4 minutes. The maximum rate of growth is reached by the eighth or ninth hour.

At $P_H$+ 5.4 the minimum generation time is 41.3 minutes. The maximum rate of growth is reached after $9\frac{3}{4}$ hours.

85 (1460)

## The pathogenicity of bacterium melitensis for guinea pigs.

By **K. F. MEYER, E. C. FLEISCHNER** and **E. B. SHAW.**

[*From the George Williams Hooper Foundation for Medical Research, University of California Medical School, San Francisco.*]

The very close relationship of *B. abortus* (Bang) to *B. melitensis* demonstrated by A. C. Evans[1] has been confirmed by our observations. Experimental studies, to be published in the near future, have furnished two additional characteristics of similarity.

1. The guinea pigs infected with *B. abortus* developed striking reactions of cutaneous hypersensitiveness with melitensis-protein, and vice versa in animals successfully infected with *B. melitensis* skin reactions are obtained with aborto-protein (see Table I.).

2. In a series of attempts to infect guinea pigs with various known strains of *B. melitensis* obtained from the Hygienic Laboratory, U. S. Public Health Service (same strains as studied by Miss Evans), we finally succeeded in producing by intratesticular injection of 1/10 agar slant (48 hours' growth on peptic digest agar) a disease with pathological changes which could not be distinguished from those seen in about one hundred guinea pigs suffering from abortion disease. The lesions of two guinea pigs, which consisted of a very large spleen, general lymphadenopathy, liver and lung lesions with infiltrations of epithelioid cells and lymphocytes were only definitely diagnosed to be the result of a *B. melitensis* infection by cross agglutination and absorption

[1] A. C. Evans, *J. Infec. Diseases*, 1918, xxii., 580.

## Table I.

| Guinea Pig. | Sex. | Wt. | Mode of Infection. | Time Interval. Days. | Cutaneous Hypersensitiveness. | | | | Time Interval. | Cutaneous Hypersensitiveness. | | | | Day on which sacrificed. | Autopsy Findings. | Bacteriologic Results. | Agglutination Test. | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Melitensis Protein, Cm. | Aborto Protein, Cm. | Bronchisepticus Protein, Cm. | Typho Protein, Cm. | | Melitensis Protein, Cm. | Aborto Protein, Cm. | Paratypho Protein, Cm. | Typho Protein, Cm. | | | | *B. abortus* | *B. melitensis.* |
| 580 | ♂ | 528 | 1/10 slant of *B. abortus* 320 and 317 interperitoneally. | 55 | 2.0<br>×<br>2.0<br>R.I.R. | 1.5<br>×<br>1.5<br>R.I.R. | 0.5<br>×<br>0.5 | 0.5<br>×<br>0.5 | 88 | NP | NP | NP | NP | 121 | Emaciation, atrophy of testes; abscess in r. epididymis; typical abortion disease. | Pus from abscess *B. abortus.* | | |
| 581 | ♀ | 489 | 1/10 slant of *B. abortus* 320 and 317 interperitoneally. | 55 | 1.3<br>×<br>1.3<br>R.I.R. | 1.7<br>×<br>1.7<br>R.I.R. | 0.6<br>×<br>0.6 | 0.5<br>×<br>0.5 | — | — | — | — | — | 63 | Abortion disease; spleen 5 × 2.6; involvement of r. metatarsophalangeal joint. | Spleen culture: *B. abortus.* | | |
| 586 | ♂ | 481 | 1/10 slant of polyvalent *B. melitensis* (1, 2, 3, 4) I.P. | 55 | 0.6<br>×<br>0.6 | 0.7<br>×<br>0.7 | 0.6<br>×<br>0.6 | 0.7<br>×<br>0.7 | 88 | 0.8<br>×<br>0.8 | 0.6<br>×<br>0.6 | 0.8<br>×<br>0.8 | 0.8<br>×<br>0.8 | 107 | Negative. | Negative. | | |
| 587 | ♂ | 550 | 1/10 slant of polyvalent *B. melitensis* (1, 2, 3, 4) I.P. | 55 | 0.9<br>×<br>0.9<br>R. | 1.0<br>×<br>1.0<br>R. | 0.4<br>×<br>0.4 | 0.5<br>×<br>0.5 | — | — | — | — | — | 63 | Negative. | Negative. | | |
| 588 | ♂ | 442 | 1/10 slant of polyvalent *B. melitensis* (1, 2, 3, 4) I.P. | 55 | 0.6<br>×<br>0.6 | 0.5<br>×<br>0.5 | 0.5<br>×<br>0.5 | 0.5<br>×<br>0.5 | — | — | — | — | — | — | Reserved for further tests. | — | | |

| Guinea Pig. | Sex. | Wt. | Mode of Infection. | Time Interval, Days. | Cutaneous Hypersensitiveness. | | | | Time Interval. | Cutaneous Hypersensitiveness. | | | | Day on which sacrificed. | Autopsy Findings. | Bacteriologic Results. | Agglutination Test. | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Melitensis Protein, Cm. | Aborto Protein, Cm. | Bronchisepticus Protein, Cm. | Typho Protein, Cm. | | Melitensis Protein, Cm. | Aborto Protein, Cm. | Paratypho Protein, Cm. | Typho Protein, Cm. | | | | *B. abortus.* | *B. melitensis.* |
| 589 | ♀ | 495 | 1/10 slant of polyvalent *B. melitensis* (1, 2, 3, 4) I.P. | 55 | 0.8<br>×<br>0.8 | 0.9<br>×<br>0.9 | 0.6<br>×<br>0.6 | 0.6<br>×<br>0.6 | 88 | 0.4<br>×<br>0.4 | 0.4<br>×<br>0.4 | 0.8<br>×<br>0.8 | 0.8<br>×<br>0.8 | 133 | Negative. | Negative. | | |
| 592 | ♂ | 590 | 1/10 slant of *B. melitensis* intratesticularly. | — | — | — | — | — | — | — | — | — | — | 10D | Lymphadenitis, abscess in r. testis; spleen enlarged. | Cultures from testis and spleen positive for *B. melitensis.* | | |
| 593 | ♂ | 558 | 1/10 slant of *B. melitensis* intratesticularly. | 55 | 1.9<br>×<br>1.9<br>R.I. | 1.1<br>×<br>1.1<br>R.I. | 0.6<br>×<br>0.6<br>C.N. | 0.5<br>×<br>0.5<br>C.N. | — | — | — | — | — | 63 | General lymphadenitis; spleen enlarged 4.5 × 2.5; surface nodular; liver foci. Striking resemblance to abortion disease. | Spleen and liver cultures positive for *B. melitensis.* | | |
| 612 | ♂ | 815 | 1/10 slant of *B. melitensis* intratesticularly. | 25 | 0.6<br>×<br>0.6 | 0.6<br>×<br>0.6 | 0.4<br>×<br>0.4 | 1.0<br>×<br>1.0 | 52 | 0.9<br>×<br>0.9 | 0.8<br>×<br>0.8 | NP | NP | 62 | Negative. | Negative. | | |

| Guinea Pig. | Sex. | Wt. | Mode of Infection. | Time Interval, Days. | Cutaneous Hypersensitiveness. | | | | Time Interval. | Cutaneous Hypersensitiveness. | | | | Day on which sacrificed. | Autopsy Findings. | Bacteriologic Results. | Agglutination Test. | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Melitensis Protein, Cm. | Aborto Protein, Cm. | Bronchisepticus Protein, Cm. | Typho Protein, Cm. | | Melitensis Protein, Cm. | Aborto Protein, Cm. | Paratypho Protein, Cm. | Typho Protein, Cm. | | | | *B. abortus.* | *B. melitensis.* |
| 613 | ♂ | 833 | 1/10 slant of *B. melitensis* intratesticularly. | 25 | 1.1<br>×<br>1.1<br>I.R. | 0.7<br>×<br>0.7 | 0.7<br>×<br>0.7 | 0.5<br>×<br>0.5 | 52 | 2.0<br>×<br>2.0<br>R.I.R. | 1.6<br>×<br>1.5<br>R.I.R. | 0.4<br>×<br>0.4 | NP | 57D | R. testis atrophic; spleen small; liver foci; emaciation. | Spleen and liver only positive. | 1 : 60–80 | 1 : 100–200 |
| 614 | ♂ | 712 | 1/10 slant of *B. melitensis* intratesticularly. | 25 | 1.5<br>×<br>1.5<br>R.I.R. | 1.2<br>×<br>1.2<br>R.I.R. | 0.8<br>×<br>0.9 | 1.0<br>×<br>1.0 | 52 | 1.5<br>×<br>1.5<br>C.N. | 1.6<br>×<br>1.6<br>C.N. | 0.7<br>×<br>0.7 | 0.7<br>×<br>0.7 | 62 | Lymphadenitis; numerous foci in liver and spleen; l. testis atrophic; abscess in epididymis; purulent seminal vesiculitis. | Cultures from urine, testis, spleen positive. | 1 : 200–800 | 1 : 1000–2000 |
| 615 | ♂ | 690 | 1/10 slant of *B. melitensis* intratesticularly. | 25 | 0.4<br>×<br>0.4 | 0.9<br>×<br>0.9<br>R.I.R. | 0.4<br>×<br>0.4 | 0.7<br>×<br>0.7 | 52 | 1.6<br>×<br>1.4<br>R.I.R. | 1.5<br>×<br>1.5<br>R.I.R. | NP | NP | 62 | Pelvic lymphadenitis; r. testis atrophic; purulent seminal vesiculitis. | Pus in semen vesicle positive. | 1 : 400 | 1 : 400–1000 |
| 617 | ♂ | 477 | 1/10 slant of *B. melitensis* intratesticularly. | 25 | 0.8<br>×<br>0.8<br>R.I. | 1.1<br>×<br>1.1<br>R.I. | 0.6<br>×<br>0.6 | 0.6<br>×<br>0.6 | 52 | 0.6<br>×<br>0.6 | 1.0<br>×<br>1.0 | NP | NP | 62 | Mediastinal lymph-node enlarged; other organs negative. | All organs negative. | 1 : 100–1 : 200 | 1 : 400–1 : 800 |
| 619 | ♂ | 445 | 1/10 slant of *B. melitensis* intratesticularly. | 25 | 1.2<br>×<br>1.2<br>R.I.R. | 1.2<br>×<br>1.2<br>R.I.R. | 0.8<br>×<br>0.8 | 0.8<br>×<br>0.8 | — | — | — | — | — | 38 | Spleen 3.5 × 2.0; general lymphadenitis; r. testis atrophic; liver and lung foci with epithelioidal cells. | Spleen only, few colonies of *B. melitensis.* | 1 : 100 | 1 : 800 |

tests with the recovered bacteria. The observations thus far completed are summarized in Table I. For comparison, the records of two guinea pigs successfully infected with the *B. abortus*, and four animals unsuccessfully inoculated intraperitoneally with *B. melitensis* are included.

Experiments with *B. melitensis* strains obtained from Algiers and with the *B. paramelitensis* (Nègre and Raynaud) are in progress.

## RECAPITULATION OF THE NAMES OF THE AUTHORS AND OF THE TITLES OF THE COMMUNICATIONS.

### VOLUME XVI.

**Amoss, H. L.**

1409. [with **H. D. Taylor** and **W. D. Witherbee.**] The effect of large doses of X-rays on the resistance of monkeys to experimental poliomyelitis.

**Baldwin, M. E.**

1387. See **Sherman, H. C.**

**Banta, Arthur M.**

1444. The results of selection with a *Cladocera* pure line (clone).

**Barber, W. Howard.**

1440. Gastric response to extragastric irritation.

**Barbour, Henry G.**

1451. The antipyretic action of dextrose.

**Barsky, Joseph.**

1395. [with **Max Kahn.**] Study of the chemistry of pernicious anemia.

**Blakeslee, A. F.**

1448. Sexual differentiation in the bread molds.

**Bridges, C. B.**

1431. See **Morgan, T. H.**

**Bronfenbrenner, J.**

1402. [with **M. J. Schlesinger.**] Carbohydrate fermentation by bacteria as influenced by the composition of the medium.

**Calkins, Gary N.**

1411. The effect of conjugation.

**Coca, Arthur F.**

1403. The mechanism of corpuscle and serum anaphylaxis in the rabbit.

**Coombs, Helen C.**

1405. See **Pike, F. H.**

**Cooper, Georgia M.**

1442. See **Park, William H.**

**Cope, Otis M.**

1432. See **Lombard, Warren P.**

**Danchakoff, Vera.**

1414. Mesenchymal activity as a factor in resistance against mouse sarcoma in chick.

**Davenport, C. B.**

1445. Exhibit showing the results of selection for a new buff race.

**Davis, Walter S.**

1383. See **Kober, Philip Adolph.**

**Dellenbaugh, Anne G.**

1388. See **Myers, Victor C.**

**Egerer, Grete.**

1382. [with **Frances Ford.**] The probable cause for the failure of some sodium tungstate to give a suitable reagent for the determination of uric acid.

**Eggleston, Cary.**

1385. [with **Robert A. Hatcher.**] Preliminary report on the behavior of certain local anesthetics.

1416. See **Hatcher, Robert A.**

**Erdmann, Rhoda.**

1412. Endomixis and size variations in pure lines of *Paramecium aurelia.*

**Fine, Morris S.**

1417. See **Myers, Victor C.**

**Fleischner, E. C.**

1460. See **Meyer, K. F.**

**Ford, Frances.**

1382. See **Egerer, Grete.**

**Gettler, A. O.**

1438. The detection of small amounts of chloral in the presence of chloroform and formalin embalming fluid.

**Gies, William J.**

1381. See **Harrow, Benjamin.**

1408. See **Lowenstein, G. A.**

**Githens, T. S.**

1378. [with **I. S. Kleiner, A. L. Meyer** and **S. J. Meltzer.**] A method of producing experimental shock.

1380. [with **I. S. Kleiner, A. L. Meyer** and **S. J. Meltzer.**] Influence of mere opening the abdomen, of state of shock, and of subsequent section of sciatic nerve upon the blood flow from the femoral vein in cats.

**Givens, Maurice H.**

1377. [with **Harry B. McClugage.**] Preliminary observations on the value of raw and dried tomatoes as antiscorbutic foods for guinea pigs.

**Harris, J. Arthur.**

1450. The transformation of the plant ovule into an ovary.

**Harrow, Benjamin.**

1381. [with **William J. Gies.**] Experimental studies of plant pigments.

**Hastings, A. Baird.**

1405. See **Pike, F. H.**

**Hatcher, Robert A.**

1385. See **Eggleston, Cary.**

1416. [with **Cary Eggleston.**] The behavior of certain digitalis principles in the body.

**Hess, Alfred F.**

1376. [with **Lester J. Unger.**] Canned tomatoes as an antiscorbutic.

1401. [with **John A. Killian.**] Chemistry of the blood in scurvy.

1407. [with **Lester J. Unger.**] The effect of heat, age and reaction on the antiscorbutic potency of vegetables.

**Holland, Dorothy F.**

1427. See **Winslow, C.-E. A.**

**Kahn, Max.**

1395. See **Barsky, Joseph.**

1453. Non-protein sulphur of the blood: Its determination, its fractionation and its clinical significance.

**Kast, Ludwig.**

1456. [with **John A. Killian.**] Some significant chemistry changes in the blood coincident with malignant tumors.

**Killian, John A.**

1400. See **Myers, Victor C.**

1401. See **Hess, Alfred F.**

1456. See **Kast, Ludwig.**

**Kleiner, I. S.**

1378. See **Githens, T. S.**

1380. See **Githens, T. S.**

**Knudson, Arthur.**

1404. See **Ottenberg, Reuben.**

**Kober, Philip Adolph.**

1383. [with **Walter S. Davis.**] A simple method for making p-arsanilic acid.

1391. A method of preparing pure dihydrochloride of diaminodioxyarsenobenzene.

**Kohman, Emma A.**

1443. A preliminary note on the experimental production of edema as related to " war dropsy."

**Kosakai, M.**

1436. The mechanism of boric acid hemolysis.

1441. The nature of osmotic pressure.

**Laughlin, H. H.**

1449. The relation between the number of chromosomes of a species and the rate of elimination of mongrel blood by the pure sire method.

**Levin, Isaac.**

1390. [with **Michael Levine.**] Malignancy of the crown gall and its analogy to animal cancer.

**Levine, Michael.**

1390. See **Levin, Isaac.**

**Little, C. C.**

1447. Some factors influencing the human sex-ratio.

**Loeb, Jacques.**

1398. Volumetric analysis of ion-protein compounds.

**Lombard, Warren P.**

1432. [with **Otis M. Cope.**] Effect of position of body on the length of systole and diastole and rate of pulse in man.

**Lowenstein, G. A.**

1408. [with **William J. Gies.**] Studies of saliva in its relation to the teeth. (1) On the normal composition of saliva. (2) Does normal saliva contain uric acid (urate)?

**Lusk, William C.**

1437. The disinfection of vitalized tissues and the healing of wounds with chinosol and salt.

**McArthur, C. G.**

1434. See **Mehrtens, H. G.**

**McClugage, Harry B.**

1377. See **Givens, Maurice H.**

**MacDougal, D. T.**

1396. [with **H. A. Spoehr.**] The effect of organic acids and their amido-compounds on the hydration of agar and on a biocolloid.

**MacDowell, E. C.**

1446. The influence of parental alcoholism upon habit formation in albino rats.

**Macht, David I.**

1392. [with **D. E. Nelson.**] On the antiseptic action of benzyl alcohol.

1393. [with **J. Weiner.**] On the action of opium alkaloids on *Trypanosoma brucei*.

1423. On the anti-spasmodic and anesthetic properties of benzaldehyde.

1424. [with **S. Matsumoto.**] A biological test for corpus luteum extracts in vitro.

1452. Concerning the effect of prostate feeding on tadpoles.

**Macleod, J. J. R.**

1394. The spontaneous development of an acidosis condition in decerebrate cats.

**MacNeal, W. J.**

1426. The influenza epidemic of 1918 in the American Expeditionary Forces in France and England.

**MacNider, William deB.**

1422. On the occurrence of degenerative changes in the liver in animals intoxicated by mercuric chloride and by uranium nitrate.

**Matsumoto, S.**

1424. See **Macht, David I.**

**Mehrtens, H. G.**

1434. [with **C. G. McArthur.**] Arsenic penetration of the meninges during the treatment of neuro-syphilis.

**Meltzer, S. J.**

1378. See **Githens, T. S.**

1380. See **Githens, T. S.**

1399. See **Wollstein, Martha.**

**Mendel, Lafayette B.**

1384. See **Osborne, Thomas B.**

1433. See **Osborne, Thomas B.**

**Meyer, A. L.**

1378. See **Githens, T. S.**

1380. See **Githens, T. S.**

**Meyer, K. F.**

1459. See **Schoenholtz, P.**

1460. [with **E. C. Fleischner** and **E. Shaw.**] The pathogenicity of bacterium melitensis for guinea pigs.

**Mohr, O. L.**

1430. [with **A. H. Sturtevant.**] A semi-lethal in *Drosophila funebris* that causes an excess of males.

**Moore, A. R.**

1397. The carbon-dioxide of injury and of respiration in nervous tissue.

1413. Electrical stimulation and $CO_2$ production in nervous tissue.

**Morgan, T. H.**

1431. [with **C. B. Bridges.**] The construction of chromosome maps.

**Myers, Victor C.**

1388. [with **Anne G. Dellenbaugh.**] Studies on the amylolytic activity of human saliva with a new method.

1400. [with **John A. Killian.**] The prognostic value of the creatinine of the blood in nephritis.

1417. [with **Morris S. Fine.**] The relative importance of the intestine and kidneys as excretory channels.

**Nelson, D. E.**

1392. See **Macht, David I.**

**Olmstead, Miriam.**

1386. A bacteriological report of influenza cases at Presbyterian Hospital.

**Osborne, Thomas B.**

1384. [with **Lafayette B. Mendel.**] Vitamines in green leaves.

1433. [with **Lafayette B. Mendel.**] The extraction of " fat-soluble vitamine " from green foods.

**Ottenberg, Reuben.**

1404. [with **Arthur Knudson.**] The effect of oxidation on Wassermann antigen.

**Palmer, Walter W.**

1455. See **Van Slyke, Donald D.**

**Pappenheimer, Alwin M.**

1428. The effects of intravenous injections of dichlorethyl-sulphide in rabbits.

**Park, William H.**

1439. Immunity results from toxin-antitoxin injections.

1442. [with **Anna W. Williams** and **Georgia M. Cooper.**] The results of the use of absorption of agglutinin on the identification of strains of influenza bacilli.

**Pike, F. H.**

1405. [with **Helen C. Coombs** and **A. Baird Hastings.**] The changes in the concentration of the carbon dioxide of the blood following changes in the circulation through the medulla oblongata.

**Salvesen, Harold A.**

1454. See **Van Slyke, Donald D.**

**Schoenholtz, P.**

1459. [with **K. F. Meyer.**] The optimum H-ion concentration for the growth of *B. typhosus* and the effect of changes in H-ion concentration on the generation time.

**Schlesinger, M. J.**

1402. See **Bronfenbrenner, J.**

**Shaw, E. B.**

1460. See **Meyer, K. F.**

**Sherman, H. C.**

1387. [with **A. W. Thomas** and **M. E. Baldwin.**] Influence of hydrogen ion concentration upon enzymic activity of three typical amylases.

**Sherwin, Carl P.**

1379. Metabolism of p-hydroxybenzoic acid and p-hydroxyphenyl acetic acid in the monkey.

**Smith, G. H.**

1410. See **Winternitz, M. C.**

**Smith, P. E.**

1418. The pigment changes in frog larvæ deprived of the epithelial hypophysis.

1419. On the reaction of the pigment cells in normal and albinous frog larvæ.

1420. Upon the experimental exchange of skin transplants between normal and albinous larvæ.

1421. On the effects of ablation of the epithelial hypophysis on the other endocrine glands.

**Spoehr, H. A.**

1396. See **MacDougal, D. T.**

**Stadie, William C.**

1425. Arterial and venous oxygen in pneumonia and influenza.

**Stockard, Charles R.**

1429. Developmental rate and the formation of embryonic structures.

**Sturtevant, A. H.**

1430. See **Mohr, O. L.**

**Taylor, C. V.**

1435. The neuro-motor system of *Euplotes*.

**Taylor, H. D.**

1409. See **Amoss, H. L.**

**Thomas, A. W.**

1387. See **Sherman, H. C.**

**Uhlenhuth, Eduard.**

1389. Parathyroids and calcium metabolism.

1406. The influence of milk upon tetany in salamander larvæ.

1415. Further proof of the antagonism existing between the thymus and parathyroid.

**Unger, Lester J.**

1376. See **Hess, Alfred F.**

1407. See **Hess, Alfred F.**

**Van Slyke, Donald D.**

1454. [with **Harold A. Salvesen.**] Determination of carbon monoxide in blood.

1455. [with **Walter W. Palmer.**] Titration of organic acids in urine.

**Warthin, Aldred Scott.**

1457. [with **Carl Vernon Weller.**] The toxic action of dichlorethylsulphide (" mustard gas ").

1458. [with **Carl Vernon Weller.**] The pathology of dichlorethylsulphide (" mustard gas ") poisoning. 83 (1458).

**Weiner, J.**

1393. See **Macht, D. I.**

**Weller, Carl Vernon.**

1457. See **Warthin, Aldred Scott.**

1458. See **Warthin, Aldred Scott.**

**Williams, Anna W.**

1442. See **Park, William H.**

**Winslow, C.-E. A.**

1427. [with **Dorothy F. Holland.**] The disinfectant action of glycerol in varying concentrations.

**Winternitz, M. C.**

1410. [with **G. H. Smith.**] Intrapulmonary irrigation.

**Witherbee, W. D.**

1409. See **Amoss, H. L.**

**Wollstein, Martha.**

1399. [with **S. J. Meltzer.**] Experimental pneumonia produced by *Streptococcus hemolyticus*.

# EXECUTIVE PROCEEDINGS.

## Main Society.

### Ninety-third Meeting.

*Cornell University Medical College, October 16, 1918. President Gies in the chair.*

*Members present:* Auer, Edwards, Funk, Gies, Githens, Givens, Hess, Killian, Kleiner, Meltzer, Meyer, A. L., Myers, Peirce, Torrey, Uhlenhuth.

### Ninety-fourth Meeting.

*New York Post Graduate Medical School, November 20, 1918. President Gies in the chair.*

*Members present:* Atkinson, Gies, Harris, Hess, Jackson, Killian, Kober, Levin, Myers, Noble, Peirce, Uhlenhuth.

### Ninety-fifth Meeting.

*Rockefeller Institute for Medical Research, December 18, 1918. President Gies in the chair.*

*Members present:* Amoss, Auer, Cole, Dochez, Edwards, Fine, Gies, Harris, Hess, Hunt, Killian, Loeb, J., Meltzer, Moore, Myers, MacDougal, Ottenberg, Peirce, Sherman, Torrey, Uhlenhuth, Wollstein.

*Members elected:* Martin Cohen, J. Arthur Harris, Mary Swartz Rose.

### Ninety-sixth Meeting.

*College of Physicians and Surgeons, January 15, 1919. President Gies in the chair.*

*Members present:* Atkinson, Auer, Coca, Edwards, Fine, Goldfarb, Hatcher, Hess, Jackson, Jobling, Killian, Levin, Lieb, Lusk, Meltzer, Myers, Ottenberg, Pike, Sherman, Thro, Uhlenhuth, Wood.

*Members elected:* Arthur F. Chace, Peter K. Olitsky, J. W. Shive.

**Ninety-seventh Meeting.**

*College of the City of New York, February 19, 1919. President Gies in the chair.*

*Members present:* Calkins, Cohen, Fine, Funk, Gies, Githens, Goldfarb, Hatcher, Hess, Jackson, Killian, Kleiner, Meltzer, Meyer, A. L., Moore, Myers, Pappenheimer, Pike, Thro, Uhlenhuth.

*Member elected:* Andrew Eggstein.

The meeting was held at 5.00 P.M., and was followed by a dinner at 7.15 P.M. Election of officers for the ensuing year occurred after the dinner and resulted as follows:

President, Gary N. Calkins; Vice-President, George B. Wallace; Secretary-Treasurer, Holmes C. Jackson; additional members of the Council, Henry C. Sherman, James W. Jobling.

**Ninety-eighth Meeting.**

*Columbia University, Schermerhorn Hall, March 19, 1919. President Calkins in the chair.*

*Members present:* Calkins, Davenport, Edwards, Gies, Githens, Hess, Jackson, MacNeal, Morgan, Myers, Oppenheimer, Pappenheimer, Ringer, Rose, Stockard, Thro, Uhlenhuth, Wallace, Williams, Anna W., Wilson.

*Member elected:* Barnett Cohen.

**Ninety-ninth Meeting.**

*University and Bellevue Hospital Medical College, April 16, 1919. President Calkins in the chair.*

*Members present:* Auer, Barber, Calkins, Gies, Jackson, Myers, Noble, Park, Ringer, Salant, Uhlenhuth.

*Members elected:* C. V. Bailey, E. Newton Harvey, Charles A. Kofoid, Arno B. Luckhardt, Herman J. Muller, H. M. Richards, Arthur H. Smith, A. H. Sturtevant, A. Waksman.

**One hundredth Meeting (Centennial Meeting).**

*Laboratory for Experimental Evolution, Cold Spring Harbor, May 24, 1919. President Calkins in the chair.*

*Members present:* Banta, Benedict, Berg, Calkins, Churchman, Davenport, DuBois, Edwards, Fine, Gager, Githens, Green-

wald, Harris, Hess, Jackson, Janney, Killian, Kirkbride, Kober, Lusk, MacNeal, Meltzer, Moore, Morgan, Mosenthal, Myers, Park, W. H., Riddle, Rose, Anton R., Rose, Mary S., Thro, Uhlenhuth, Wallace, Williams, Anna W.

At the close of the scientific program, the members of the Society were the luncheon guests of Dr. Graham Lusk, at his home in Cold Spring Harbor. A business meeting was held subsequent to the luncheon at which the following members were elected:

Albert F. Blakeslee, W. R. Bloor, Robert Chambers, H. H. Laughlin, C. C. Little, E. Carleton MacDowell, Charles W. Metz, Philip E. Smith.

Informal talks were given by Dr. S. J. Meltzer, Dr. Graham Lusk, Dr. George B. Wallace and Dr. C. B. Davenport.

The meeting then adjourned.

## PACIFIC COAST BRANCH.

### Twenty-first Meeting.

*San Francisco, California, March 5, 1919.*

*Members present:* Alvarez, Burnett, Evans, Gay, Hewlett, Ophüls, Schmidt, Walker, Whipple.

### Twenty-second Meeting.

*Berkeley, California, May 15, 1919.*

*Members present:* Burnett, Crawford, Evans, Gay, Ophüls, Schmidt, Walker, Whipple.

# REGISTER OF NAMES AND ADDRESSES OF THE MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

ABBOTT, ALEXANDER C.....University of Pennsylvania.
ABEL, JOHN J.....Johns Hopkins University.
ADAMI, J. GEORGE.....McGill University, Montreal.
ADDIS, THOMAS.....Leland Standford University, San Francisco.
ADLER, HERMAN M.....Juvenile Psychopathic Institute, Chicago.
ALLEN, A. REGINALD.....University of Pennsylvania.
ALLEN, BENNET M.....University of Kansas.
ALSBERG, CARL L.....U. S. Department of Agriculture, Washington, D. C.
ALVAREZ, WALTER C.....University of California.
AMOSS, HAROLD L.....Rockefeller Institute for Medical Research.
ANDERSON, JOHN F.....New Brunswick, N. J.
ATKINSON, JAMES P.....Department of Health, New York City.
AUER, JOHN.....Rockefeller Institute for Medical Research.
AUSTIN, J. H.....University of Pennsylvania.

BAEHR, GEORGE.....Mt. Sinai Hospital, N. Y. City.
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*Cleveland, Ohio, 8803 Euclid Ave.*—Katharine R. Collins.
*Kastanienbaum, Switzerland.*—Fritz Schwyzer.
*New Brunswick, N. J.*—John F. Anderson, Isaac F. Harris.
*Otisville, N. Y.*—B. White.
*Rosebank, N. Y.*—Oscar Teague.
*Silver City, New Mexico.*—E. M. Ewing.
*Upper Montclair, N. J.*—George Peirce.
*Washington, D. C., 3315 Wisconsin Ave.*—C. J. West.
*Winnipeg, Canada.*—F. J. Birchard.
*Germany.*—Beutner, Reinhard.

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**Members present at the ninety-fifth meeting:**

Amoss, Auer, Cole, Dochez, Edwards, Fine, Gies, Harris, Hess, Hunt, Killian, Loeb, J., Meltzer, Moore, Myers, V. C., MacDougal, Ottenberg, Peirce, Sherman, Torrey, Uhlenhuth, Wollstein.

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**Members elected at the ninety-fifth meeting:**

Martin Cohen, J. Arthur Harris, Mary Swartz Rose.

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**Dates of the next two meetings:**

January 15, 1919–February 19, 1919.

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*Fordham University School of Medicine.*— A. O. Shaklee, Carl P. Sherwin.

*Hospitals, Bellevue.*—Charles Norris. *Beth Israel.*—Max Kahn. *Memorial Hospital.*—H. H. Janeway. *Montefiore Home.*—George Fahr, Nelson W. Janney, B. S. Kline. *Mt. Sinai.*—George Baehr, Charles A. Elsberg, Albert A. Epstein, R. Ottenberg, Harry Plotz, M. A. Rothschild. *New York.*—A. F. Coca. *Presbyterian.*—Ralph A. Kinsella, Miriam P. Olmstead, Walter W. Palmer. *Roosevelt.*—K. G. Falk, I. Greenwald, W. L. Lyle. *St. Lukes.*—L. W. Famulener.

*New York City Departments. Education.*—C. Ward Crampton. *Health.*—James P. Atkinson, Edwin J. Banzhaf, C. B. Fitzpatrick, Alfred F. Hess, Anna W. Williams, A. Zingher.

*New York Post-Graduate Medical School.*—Ludwig Kast, John A. Killian, W. J. MacNeal, V. C. Myers, Anton R. Rose, R. M. Taylor.

*New York University.*—W. H. Barber, Harlow Brooks, Warren Coleman, J. W. Draper, Edward K. Dunham, A. O. Gettler, E. R. Hoskins, Holmes C. Jackson' Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D. Senior, Douglas Symmers, George B. Wallace.

*Rockefeller Institute for Medical Research.*—Harold L. Amoss, John Auer, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole, A. R. Dochez, Simon Flexner, F. L. Gates, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Louise Pearce, Peyton Rous, Edward Uhlenhuth, Donald D. Van Slyke, Martha Wollstein.

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## Non-Resident.

*Agricultural Experiment Stations. Connecticut (New Haven).*—Thomas B. Osborne. *Maine (Orono).*—Raymond Pearl. *Maryland (Beltsville).*—E. B. Meigs.

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*Otisville, N. Y.*—B. White.
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*Winnipeg, Canada.*—F. J. Birchard.
*Germany.*—Beutner, Reinhard.

Members present at the ninety-fourth meeting:

Atkinson, Gies, Harris, Hess, Jackson, Killian, Kober, Levin, Myers, Noble, Peirce, Uhlenhuth.

## Dates of the next two meetings:

December 18, 1918–January 15, 1919

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**Dates of the next two meetings:**

November 20, 1918–December 18, 1918

# PROCEEDINGS

OF THE

# SOCIETY FOR

# EXPERIMENTAL BIOLOGY AND MEDICINE

NINETY-SIXTH MEETING

COLLEGE OF PHYSICIANS AND

SURGEONS

JANUARY 15, 1919

NEW YORK CITY

VOLUME XVI

No. 4

NEW YORK

1919

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OF THE

# SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

NINETY-SEVENTH MEETING

COLLEGE OF THE CITY OF NEW YORK

FEBRUARY 19, 1919

NEW YORK CITY

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NINETY-EIGHTH MEETING

SCHERMERHORN HALL

COLUMBIA UNIVERSITY

MARCH 19, 1919

NEW YORK CITY

AND

TWENTY-FIRST MEETING PACIFIC COAST BRANCH,

SAN FRANCISCO, CALIFORNIA

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Barnett Cohen.

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OF THE

# SOCIETY FOR

# EXPERIMENTAL BIOLOGY AND MEDICINE

NINETY-NINTH MEETING

UNIVERSITY AND BELLEVUE HOSPITAL

MEDICAL COLLEGE

NEW YORK CITY

APRIL 16, 1919

VOLUME XVI

No. 7

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1919

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ONE HUNDREDTH MEETING

LABORATORY FOR EXPERIMENTAL
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COLD SPRING HARBOR
NEW YORK
MAY 24, 1919

AND

TWENTY-SECOND MEETING
PACIFIC COAST BRANCH
BERKELEY, CALIFORNIA
MAY 15, 1919

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No. 8

NEW YORK
1919

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*Fordham University School of Medicine.*— A. O. Shaklee, Carl P. Sherwin.

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*New York Post-Graduate Medical School.*—Arthur F. Chace, Martin Cohen, Ludwig Kast, John A. Killian, W. J. MacNeal, V. C. Myers, Anton R. Rose, R. M. Taylor.

*New York University.*—W. H. Barber, Harlow Brooks, Warren Coleman, J. W. Draper, Edward K. Dunham, A. O. Gettler, Holmes C. Jackson, Arthur R. Mandel, John A. Mandel, W. C. Noble, William H. Park, H. D . Senior, Douglas Symmers, George B. Wallace.

*Rockefeller Institute for Medical Research.*—Harold L. Amoss, John Auer, Wade H. Brown, C. G. Bull, Alexis Carrel, A. E. Cohn, Rufus Cole, A. R. Dochez, Simon Flexner, F. L. Gates, T. S. Githens, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Peter K. Olitsky, Louise Pearce, Peyton Rous, Eduard Uhlenhuth, Donald D. Van Slyke, Martha Wollstein.

*Brooklyn Botanic Garden.*—C. Stuart Gager, O. E. White.

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893 *Park Ave., N. Y. City.*—Walter L. Niles.

141 *W. 78th St., N. Y. City.*—A. I. Ringer.

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## Non-Resident.

*Agricultural Experiment Stations. Connecticut* (*New Haven*).—Thomas B. Osborne. *Maryland* (*Beltsville*).—E. B. Meigs. *New Jersey* (*New Brunswick*).—J. W. Shive.

*Carnegie Institution of Washington.* (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*).—A. M. Banta, Charles B. Davenport, J. Arthur Harris, O. Riddle. (*Desert Laboratory, Tucson, Ariz.*).—D. T. MacDougal. (*Tortugas, Florida*).—Alfred G. Mayer.

*State Boards of Health. New York* (*Albany*).—Mary B. Kirkbride, P. A. Kober, A. B. Wadsworth.

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*Institutes. Gratwick Laboratory (Buffalo).*—G. H. Clowes, H. R. Gaylord. *Juvenile Psychopathic (Chicago).*—Herman M. Adler. *Phipps (Philadelphia).*—Paul A. Lewis. *Rockefeller (Princeton).*—F. S. Jones, Theobald Smith, Paul E. Howe, Carl Ten Broeck. *Wistar (Philadelphia).*—H. H. Donaldson, Shinkishi Hatai.

*U. S. Departments. Agriculture (Washington, D. C.).*—Carl L. Alsberg, William N. Berg, J. R. Mohler. *Department Laboratory (Atlanta, Ga.).*—R. L. Kahn. *Medical Corps (Fort Sam Houston, Texas).*—J. F. Siler. *Ohio River Investigation (Ohio).*—W. H. Frost.

*Universities. Buffalo.*—Herbert U. Williams. *California.*—Walter C. Alvarez, T. C. Burnett, George W. Corner, H. M. Evans, F. P. Gay, S. J. Holmes, Charles H. Hooper, Samuel H. Hurwitz, R. A. Kocher, W. P. Lucas, S. S. Maxwell, K. F. Meyer, Carl L. A. Schmidt, E. L. Walker, C. K. Watanabe, G. H. Whipple. *Chicago.*—A. J. Carlson, Frank R. Lillie, Shiro Tashiro, H. Gideon Wells. *Cincinnati.*—D. E. Jackson. *Clark.*—Ralph S. Lillie. *Copenhagen.*—Christen Lundsgaard. *Cornell.*—Sutherland Simpson. *Georgia.*—Richard V. Lamar. *George Washington.*—W. H. Schultz, *Harvard.*—Jacob Bronfenbrenner, Walter B. Cannon, W. T. Councilman, Harvey Cushing, Otto Folin, Worth Hale, Reid Hunt, W. J. V. Osterhout, G. H. Parker, F. Pfaff, W. T. Porter, Joseph H. Pratt, M. J. Rosenau, E. E. Southard, Percy G. Stiles, Richard P. Strong, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes. *Illinois.*—H. B. Lewis, William H. Welker. *Iowa.*—Louis Baumann. *Japan.*—Noahidé Yatsu. *Jefferson.*—O. Bergeim, P. B. Hawk. *Johns Hopkins.*—John J. Abel, Walter E. Dandy, W. W. Ford, W. S. Halsted, William H. Howell, John Howland, H. S. Jennings, Walter Jones, David I. Macht, Adolph Meyer, H. O. Mosenthal, E. A. Park, Raymond Pearl, William H. Welch. *Kansas.*—Bennet M. Allen, George B. Roth. *Leland Stanford.*—Thomas Addis, George D. Barnett, A. C. Crawford, E. C. Dickson, Harold K. Faber, A. W. Hewlett, V. L. Kellogg, W. H. Manwaring, W. Ophüls, R. E. Swain. *Maryland.*—Charles E. Simon. *McGill (Montreal).*—J. George Adami, John L. Todd. *Michigan.*—C. W. Edmunds, Nellis B. Foster, G. Carl Huber, Warren P. Lombard, Frederick G Novy, Aldred S. Warthin, Carl Vernon Weller. *Minnesota.*—R. A. Gortner, A. D. Hirschfelder, E. P. Lyon, F. W. Schlutz. *Missouri.*—Mazÿck P. Ravenel. *Nebraska.*—A. E. Guenther. *North Carolina.*—W. deB. MacNider. *Northwestern.*—R. G. Hoskins. *Oregon.*—C. F. Hodge. *Peking Union Medical.*—Franklin C. McLean. *Pennsylvania.*—Alexander C. Abbott, Alfred Reginald Allen, J. H. Austin, D. H. Bergey, J. A. Kolmer, E. B. Krumbhaar, Richard M. Pearce, O. H. Perry Pepper, Edward T. Reichert, Alfred N. Richards, J. Edwin Sweet, A. E. Taylor. *Pittsburgh.*—C. C. Guthrie, W. L. Holman, E. R. Hoskins, Oskar Klotz, J. W. McMeans. *Philippine.*—R. B. Gibson. *Princeton.*—Edwin G. Conklin. *Rochester.*—Maurice H. Givens, John R. Murlin. *Rutgers.*—F. E. Chidester, A. R. Moore. *Sheffield.*—J. B. Leathes. *Southern California (Los Angeles).*—Lyman B. Stookey. *St. Louis.*—Don R. Joseph. *Toronto.*—A. H. Caulfeild, J. G. Fitzgerald, A. Hunter, A. B. Macallum, T. Brailsford Robertson. H. Wasteneys, *Tulane.*—Charles W. Duval. *Union University (Albany Medical College).*—Melvin Dresbach, Arthur Knudson. *Vanderbilt (Nashville).*—W. F. Petersen, B. T. Terry. *Virginia.*—H. E. Jordan. *Washington (St. Louis).*—M. T. Burrows, J. V. Cooke, Joseph Erlanger, Leo Loeb, Eugene L. Opie, G. C. Robinson, Philip A. Shaffer. *Wisconsin.*—Charles R Bardeen, C. H. Bunting, L. J. Cole, P. F. Clark, J. A. E. Eyster, Arthur S. Loevenhart. *Western Reserve (Cleveland).*—George W. Crile, A. B. Eisenbrey, Paul J. Hanzlik, H. T. Karsner, J. J. R. Macleod, David Marine, Torald Sollmann, G. N. Stewart. *Wisconsin.*—William Salant. *West Virginia.*—Withrow Morse. *Yale.*—George A. Baitsell, Henry Gray Barbour, R. H. Chittenden, J. W. Churchman, Rhoda Erdmann, Ross G. Harrison, Davenport Hooker, Henry Laurens, Lafayette B. Mendel, A. L. Prince, Leo F. Rettger, Reynold Albrecht Spaeth, Frank P. Underhill, C.-E. A. Winslow, Lorande Loss Woodruff.

*Amherst, Mass.*—Otto C. Glaser.
*Calco Chemical Co., Bound Brook, N. J.*—M. S. Fine.
*Cambridge, England.*—C. G. L. Wolf.
*Chicago, Illinois, 104 S. Michigan Ave.*—Solomon Strouse.
*Cleveland, Ohio, 8803 Euclid Ave.*—Katharine R. Collins.
*Kastanienbaum, Switzerland.*—Fritz Schwyzer.
*New Orleans, La.*—E. M. Ewing.
*New Brunswick, N. J.*—John F. Anderson, Isaac F. Harris.
*Otisville, N. Y.*—B. White.
*Rosebank, N. Y.*—Oscar Teague.
*Washington, D. C., 3315 Wisconsin Ave.*—C. J. West.
*Winnipeg, Canada.*—F. J. Birchard.
*Germany.*—Reinhard, Beutner.

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**Members present at the ninety-ninth meeting:**

Auer, Barber, Calkins, Gies, Jackson, Myers, V. C., Noble, Park, Ringer, Salant, Uhlenhuth.

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**Members elected at the ninety-ninth meeting:**

C. V. Bailey, E. Newton Harvey, Charles A. Kofoid, Arno B. Luckhardt, Herman J. Muller, H. M. Richards, Arthur H. Smith, A. H. Sturtevant, A. Waksman.

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**Dates of the next two meetings:**

May 24, 1919–Oct. 15, 1919.

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*Cornell University Medical College.*—Harold Bailey, Stanley R. Benedict, Robert Chambers, E. F. DuBois, D. J. Edwards, C. Eggleston, William J. Elser, James Ewing, J. A. Hartwell, Robert A. Hatcher, Graham Lusk, John R. Murlin, Charles R. Stockard, W. C. Thro, John C. Torrey.

*Fordham University School of Medicine.*—A. O. Shaklee, Carl P. Sherwin.

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*New York University.*—W. H. Barber, Harlow Brooks, Warren Coleman, J. W. Draper, Edward K. Dunham, A. O. Gettler, Holmes C. Jackson, Arthur R. Mandel, John A. Mandel, W. C. Noble, H. D. Senior, Douglas Symmers, George B. Wallace.

*Rockefeller Institute for Medical Research.*—Harold L. Amoss, John Auer, Wade H. Brown, Alexis Carrel, A. E. Cohn, Rufus Cole, A. R. Dochez, Simon Flexner, F. L. Gates, Walter A. Jacobs, I. S. Kleiner, P. A. Levene, Jacques Loeb, S. J. Meltzer, Arthur L. Meyer, Gustave M. Meyer, James B. Murphy, Hideyo Noguchi, J. H. Northrop, Peter K. Olitsky, Louise Pearce, Peyton Rous, Arthur H. Smith, Eduard Uhlenhuth, Donald D. Van Slyke, Martha Wollstein.

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141 *W. 78th St., N. Y. City.*—A. I. Ringer.

302 *Convent Ave., N. Y. City.*—William Salant.

50 *Vanderbilt Ave., N. Y. City.*—Benjamin White.

61 *East 86th St., N. Y. City.*—Louis Baumann.

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## Non-Resident.

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*Carnegie Institution of Washington.* (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*).—A. M. Banta, Albert F. Blakeslee, Charles B. Davenport, J. Arthur Harris, H. H. Laughlin, C. C. Little, E. Carleton MacDowell, Charles W. Metz, O. Riddle. (*Desert Laboratory, Tucson, Ariz.*).—D. T. MacDougal. (*Washington, D. C.*).—Alfred G. Mayer.

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*Hospitals. General* (*Cincinnati, Ohio*).—Martin H. Fischer. *Cottage* (*Santa Barbara, Calif.,*) Nelson W. Janney, *Lakeside* (*Cleveland, Ohio*).—H. T. Karsner. *Massachusetts General* (*Boston*).—David L. Edsall. *Mercy* (*Pittsfield, Mass.*).—Thomas Flournoy. *Peter Bent Brigham* (*Boston*).—Harvey Cushing, Francis W. Peabody. *Presbyterian* (*Phila.*).—Ralph Pemberton. *Robert Brigham* (*Boston*).—F. M. McCrudden. *Royal Victoria* (*Montreal*).—Horst Oertel. *St. Francis* (*Pittsburgh*).—J. W. McMeans. *Western Pennsylvania* (*Pittsburgh*).—Maurice H. Givens, Jacob Rosenbloom.

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*U. S. Departments. Agriculture* (*Washington, D. C.*).—Carl L. Alsberg. *Bureau of Animal Industry.*—William N. Berg, J. R. Mohler. *Department Laboratory* (*Atlanta, Ga.*).—R. L. Kahn. *Hygienic Laboratory* (*Washington, D. C.*).—George B. Roth. *Medical Corps* (*Fort Sam Houston, Texas*).—J. F. Siler. *Ohio River Investigation* (*Ohio*).—W. H. Frost.

*Universities. Adelaide* (*South Australia*).—T. Brailsford Robertson. *Buffalo.*—Katharine R. Collins. Herbert U. Williams. *California.*—Walter C. Alvarez, W. R. Bloor, T. C. Burnett, H. M. Evans, F. P. Gay, S. J. Holmes, Charles W. Hooper, Samuel H. Hurwitz, R. A. Kocher, Charles A. Kofoid, W. P. Lucas, S. S. Maxwell, K. F. Meyer, Carl L. A.

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*Amherst, Mass.*—Otto C. Glaser.
*Baltimore, Md.*, 1421 *Edmondson Ave.*—J. A. F. Pfeiffer.
*Bronxville, N. Y.*—Isaac F. Harris.
*Calco Chemical Co., Newark, N. J.*—M. S. Fine.
*Cambridge, England.*—C. G. L. Wolf.
*Chicago, Illinois*, 104 *S. Michigan Ave.*—Solomon Strouse, 31 *East Elm St.*—W. F. Petersen.
*Kastanienbaum, Switzerland.*—Fritz Schwyzer.
*Maceo, Kentucky.*—R. M. Taylor.
*New Orleans, La.*—E. M. Ewing.
*New Brunswick, N. J.*—John F. Anderson.
*Ocean Beach, California*, 4952 *Santa Monica.*—A. J. Carlson.
*Philadelphia, Pa.*, 1524 *Chestnut St.*—T. S. Githens.
*Rosebank, N. Y.*—Oscar Teague.
*Washington, D. C.*, 3315 *Wisconsin Ave.*—C. J. West, 1445 *Rhode Island Ave.*—Edward B. Meigs, 1864 *Park Road.*—Robert M. Yerkes.
*Winnipeg, Canada.*—F. J. Birchard.
*Germany.*—Reinhard Beutner.
*London, England.*—Cameron V. Bailey.

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**Members present at the one-hundredth meeting:**

Banta, Benedict, Berg, Calkins, Churchman, Davenport, DuBois, Edwards, Fine, Gager, Githens, Greenwald, Harris, Hess, Jackson, Janney, Killian, Kirkbride, Kober, Lusk, MacNeal, Meltzer, Moore, Morgan, Mosenthal, Myers, Park, W. H., Riddle, Rose, Anton R., Rose, Mary S., Thro, Uhlenhuth, Wallace, Williams, Anna W.

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**Members present at the twenty-second meeting of the Pacific Coast Branch:**

Burnett, Crawford, Evans, Gay, Ophüls, Schmidt, Walker, Whipple.

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**Members elected at the one-hundredth meeting:**

Albert F. Blakeslee, W. R. Bloor, Robert Chambers, H. H. Laughlin, C. C. Little, E. Carleton MacDowell, Charles W. Metz, Philip E. Smith.

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**Dates of the next two meetings:**

October 15, 1919–November 19, 1919.

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